EXHIBIT 24

UNITED STATES DISTRICT COURT

DISTRICT OF VERMONT

Civil Action Number: 5:16-cv-0125

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JAMES D. SULLIVAN, ET AL, INDIVIDUALLY AND ON BEHALF OF A CLASS OF PERSONS SIMILARLY SITUATED,

Plaintiffs,

vs

SAINT-GOBAIN PERFORMANCE PLASTICS CORPORATION,

Defendant.

****

DEPOSITION OF PHILIPPE GRANDJEAN, a witness called on behalf of the respective party, taken pursuant to the applicable provisions of the Federal Rules of Civil Procedure, before William M. Jackson, Professional Court Reporter and Notary Public in and for the Commonwealth of Massachusetts, at the Law Offices of Sugarman and Sugarman, 800 Boylston Street, Boston, Massachusetts, on October 10, 2018, commencing at 9:08 a.m.

APPEARANCES:

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I N D E X

E X H I B I T S

PROCEEDINGS

THE VIDEOGRAPHER: Good morning. We are going on the record at 9:08 a.m. on October 10, 2018. Please note microphones are sensitive and may pickup private conversations and cell phone interference. Please turn off all cellphones and place them away from the microphones. Audio and video recording will continue unless all parties agree to go off the record. This is Media Unit 1 of the Video Recorded Deposition of Philippe Grandjean in the matter of James D. Sullivan, et al versus Saint-Gobain Performance Plastics Corporation in the United States District Court, District of Vermont. 516-CV-00125.

This deposition is being held at Sugarman and Sugarman, 800 Boylston Street, Boston, Massachusetts. My name is Gail Ashton from the firm Veritext. I am the videographer. The court reporter is William Jackson from the firm Veritext. I am not authorized to administer an oath. I am not related to any party in this action nor am I financially interested in the outcome.

Now counsel and all present in the room will state their appearances and affiliations for the record.

MR. WOLFF: Bert Wolff for the Defendant.

MS. PASSARETTI-WU: Rachel Passaretti-Wu for the Defendant.

MR. WHITLOCK: James Whitlock on behalf of the Plaintiffs.

PHILIPPE GRANDJEAN

a witness called on behalf of the respective party, having been sufficiently identified and sworn to tell the truth, was examined and testified as follows:

DIRECT EXAMINATION

BY MR. WOLFF:

Q. Please state your name.

A. My name is Philippe Grandjean.

Q. As a general principle, you would agree that physicians and scientific investigators should try to look at issues critically?

A. That sounds reasonable.

Q. And physicians and scientific investigators are concerned about being accurate, true?

MR. WHITLOCK: Objection to the form.

A. I can say this much as a physician and scientist. I myself is trying to be as accurate as possible.

Q. And as a physician and scientific investigator, do you subscribe to the principle that it is important to use accuracy and precision in your writings?

A. In my writings, yes. In my writings, in this opinion for example, yes.

Q. Do you believe that scientists should describe their methods and explain their reasoning so others can understand how the data were analyzed and how the conclusions were reached?

MR. WHITLOCK: Objection to the form.

A. This is a little too general. Because we cannot start with the principles of physics. Before we do anything. In general terms, if we are, let's say, using innovative methods, this is particularly important.

Q. You would agree that criticism and rigorous attempts at refutation of a hypothesis being advanced is an integral part of the scientific method; is that correct?

MR. WHITLOCK: Objection to the form.

A. That was a very long sentence. I need it again.

Q. You would agree criticism and vigorous attempts at refutation of the hypothesis being advanced is an integral part of the scientific method, correct?

A. I would say this is in accordance with Popper's Philosophy. The positivism that it is impossible to approve usually is impossible to approve a hypothesis a hundred percent. But if we fail repeatedly, on refuting

it, that is support for the validity of that hypothesis. That is how I would state Popper's philosophy. That is part of modern science.

MR. WOLFF: Mark this as Exhibit Number 1.

(Report was marked as Exhibit Number 1 for identification)

Q. Exhibit Number 1 is a copy of your report in this matter dated August 1, 2018; is that correct?

A. That's sort of what it looks like.

Q. In the first paragraph, you write that you have been asked by counsel for the plaintiffs to provide from a medical and epidemiological perspective an expert rebuttal report responding to certain opinions authored by three experts retained by Defendant Saint-Gobain and that specifically you have been asked to provide an evaluation of the human health risks associated with environmental PFOA contamination from Saint-Gobain's manufacturing and disposal operation in North Bennington and Bennington, Vermont; is that correct?

A. That's what it says here right on Page 1 on the top.

Q. On Page 3 of your report, you further state that you have also reviewed the reports of the three experts retained by Saint-Gobain to respond by Doctor Ducatman;

is that correct?

A. That's correct.

Q. You also state that you have benefited from access to and review of Doctor Ducatman's two expert reports as well as his expert rebuttal report; is that correct?

A. That's correct.

Q. On Page 5 of your report, you state that you concur with Doctor Ducatman's opinion concerning a medical monitoring program; is that correct?

A. Is that what it says here?

Q. In the first bullet point here.

A. Yes. That a medical monitoring is appropriate. Yes.

Q. Ultimately, on Page 71 of your report in Paragraph Number 9, you assert that Doctor Ducatman's opinions are justifiable and reasonable on the basis of current scientific and medical evidence; is that correct?

A. That's correct.

Q. What is your understanding of your role in this litigation? In other words, how do you define your role here?

A. This is exactly the wording that you just read

out loud from Page 1. The top paragraph. That, I, medical and epidemiological expert to help rebut the three expert rebuttals from the defendant.

Q. Is it fair to say that you have also been asked to endorse the opinions of Doctor Ducatman in this case?

A. Not precisely. Because that was not my task. But indirectly, I certainly have relied upon his opinion, and I think if you compare the reports, we are very much in agreement.

Q. Are you aware that Doctor Ducatman was deposed in this matter?

A. I think I heard so. I have not, I don't know about the details.

Q. Because I noticed that you did not list Doctor Ducatman's deposition transcript among the materials that you considered. So have you seen that before?

A. What's that -- no. He was deposed recently. It must have been something way back. I don't remember frankly.

MR. WOLFF: Mark this as Exhibit Number 2.

(Doctor Ducatman's Deposition Transcript was marked as Exhibit Number 2 for identification).

Q. Exhibit 2.

A. I don't remember that one.

A. Exhibit Number 2 is a copy of Doctor Ducatman's deposition transcript dated February 28, 2018. Would you please turn with me to Pages 37 to 39 beginning at Line 21.

A. Repeat that.

Q. 37. Line 21. I want to ask you some questions about the next two pages.

A. Okay.

Q. So you are free to sort of peruse the transcript and look along. I want to be clear that I am asking you for your own opinions. This is not a reading exercise.

A. Right. It is 200 something pages.

Q. At this point, I am only asking you about two of them.

A. Okay.

Q. Do you agree that different people drink different amounts of water on average over the course of a day?

MR. WHITLOCK: Objection to the form.

A. This agrees with EPA exposure perimeters. I would say this is true.

Q. Do you agree that some people will typically drink only tap water, some people will typically drink only bottled water, and some people will drink a

combination of both?

MR. WHITLOCK: Objection to the form.

A. I think it is difficult only to drink bottled water, because you tend to use tap water, let's say, for boiling potatoes or making gravy. It would be hard to avoid completely trying to use tap water. I don't think that is what EPA is assuming in their exposure tables.

Q. In terms of, if we put cooking to the side, because I will ask separate questions about cooking, do you agree in terms of drinking in terms of a glass of water, some people will typically drink only tap water, some people will drink only bottled water, and some people will drink a combination of both?

MR. WHITLOCK: Objection to the form.

A. Again, I don't think this is likely that you can completely avoid tap water. Again, I am referring to EPA's tables, and I don't think they include people drinking absolutely zero water, because you still have to make tea and coffee. It would be highly unusual for people who make tea with bottled water.

Q. The sources of tap water even at areas at issue in this matter can be different from house to house?

A. I would assume so based on -- let me hear the question again.

Q. The sources of tap water even in the areas at issue in this matter can be different from house to house, true?

A. So the sources -- are you talking about the PFOA water?

Q. I am talking about where the water service comes from in the home.

A. So whether it is community water or well water, is that what you are asking?

Q. Yes.

A. Yes. Okay. I understand that's true. That's true from the map of the community.

Q. Different people will use different sources of water for cooking; is that correct?

A. Again, what do you mean sources? Are you talking if they have pipe water in the house from outside or if they have their own well? Is that what you are referring to?

Q. Some people will use tap water. Some people will use water that has been filtered with activated charcoal. Some people will use bottled water. Is that correct?

MR. WHITLOCK: Objection to the form. Calls for speculation. You may answer.

A. I can't answer that. I don't know which houses have filters. So this is not part of my report. I can't guess.

Q. Some people will cook exclusively or primarily with tap water whereas other people will cook exclusively or primarily with bottled water; is that correct?

MR. WHITLOCK: Same objection. Speculation.

A. It would be highly unusual to prepare food with bottled water. I have no knowledge of that existing in this community.

Q. One person might drink on average a glass of tap water a day whereas another person might drink on average a gallon of tap water a day; is that correct?

A. I don't know if one glass is a good problem anymore. Again, I think we need to refer to EDA's exposure tables and I think a gallon is a little bit on the high side unless it is in the far south. Again, a glass is very little. So these are really extremes.

Q. There is a continuum of the amount of water that an average person will drink over the course of a day; is that correct?

A. I would agree to that.

Q. One person might typically drink only bottled

water as a general rule; is that correct?

A. Again, I think you are really pressing the issue that I can't say that this is correct. I would say it is unlikely that a person would simply only drink bottled water. Because I refer to my comment before on tea and coffee.

Q. Some people will routinely use an activated carbon based water filter before using tap water where others might not, correct?

MR. WHITLOCK: Objection to the form. Foundation.

A. I could say yes, but I don't know if that applies to this community.

Q. Please, turn with me to Page 69 of your report. In the context of addressing medical monitoring, halfway down the page, you state that every individual has his or her own background risk of developing the diseases in question; is that correct?

A. That is what it says here.

Q. And you further state that early life exposures to PFOA will likely lead to greater risks as will exposures occurring during pregnancies or at peak ages for say cancer development?

A. That's correct.

Q. And you also state that age should be considered; is that correct?

A. Right. But look at it in this connection.

Q. Are you done with your answer?

A. Yes.

Q. I was not sure.

A. This is part of a whole paragraph. Yes.

Q. Due to obvious differences in gender and physiology, you note that monitoring blood pressure during pregnancy would not be needed for male residents screening for prostate cancer is not needed for females; is that correct?

A. Exactly.

Q. Please turn with me to Page 55 of Doctor Ducatman's deposition transcript. Beginning at the very bottom of the page at Line 25. Then carrying on to Page 61. Are you there?

A. Among the individuals?

Q. Yes.

A. Yes.

Q. Let's briefly explore whether you and Doctor Ducatman are on the same page and are in agreement with one another. Again, while you are free to read along, I am asking you whether you also share these views. Okay?

A. Yes.
Q. Among the individuals residing within the areas at issue in this matter, would you expect there to be considerable individual differences as to their amount and length of exposures to PFOA?
A. That's true. I agree.
Q. Would you expect there to be individual differences as to what their blood PFOA serum levels would be?
A. Yes. That is reasonable. I agree.
Q. Would you expect there to be individual differences as to what their susceptibilities if any to PFOA might be?
MR. WHITLOCK: Objection to the form. Susceptibilities. Being vague and ambiguous.
A. If I may just refer to my own writing. Let me explain my understanding of this. That susceptibility relates to men not developing hypertension during pregnancy and issues like that I explained in my paragraph that you read before.
Q. As to those individual differences in exposure blood levels and susceptible if any would that be a function of a number of different variables?
MR. WHITLOCK: Objection to the form.

Vague. Ambiguous.

A. It is a very convoluted sentence. It is not as precise as I would explain it in science but it seems reasonable in my understanding of it.

Q. Let's see if we can drill down on that. Among those individual differences would be a function of among other things their ages; is that correct?

A. Function of duration?

Q. Ages.

A. Yes.

Q. Among those individual differences would be a function of among other things gender, true?

A. Yes.

Q. Those individual differences would be a function of among other things their physiology; is that correct?

A. What do you mean by physiology?

Q. Well, physiology, the makeup of their body.

A. I can't answer that.

Q. The individual differences would be a function of among other things how long they lived in the area; is that true?

A. Did you say differences in exposure?

Q. Yes. Among the individual differences in exposure, blood levels and susceptibilities would be how

long they lived in the area, true?

A. I don't like the word susceptibility, because I don't understand your --

Q. Let's do it this way. Among the considerable individual differences in PFOA, exposure and blood levels, those individual differences would be a function of among other things how long they lived in the area, true?

A. In that meaning, I understand, it is true.

Q. Among the individual differences and exposure and blood levels for PFOA, those individual differences would be a function of among other things their rates of daily water consumption; is that true?

A. I agree.

Q. Among the considerable individual differences in exposure to PFOA and blood levels of PFOA, the individual differences would be a function of among other things the concentrations of PFOA in the water that they drank; is that correct?

A. That's correct.

Q. And those individual differences would be a function of among other things their sources of water; is that correct?

MR. WHITLOCK: Objection to the form.

A. What do you mean by sources?
Q. In other words, where they take the water from. If they are drinking tap water, bottled water, if they are having water from activated carbon filter systems.
A. Yes. Okay. I mean, it is so easy to understand, well, any way, my answer is yes.
Q. Among the individual differences, that would also be a function of their diet and nutrition; is that correct?
A. I would not say yes, because I am not sure how that would affect their uptake of PFOA or their exposure unless this is somebody eating soup every day. So I can't answer that.
Q. Among the considerable individual differences and susceptibilities if any to PFOA exposure, those individual differences would be a function among other things one's diet and nutrition; is that correct?
MR. WHITLOCK: Objection to the term considerable.
A. I can't answer that.
Q. The individual differences would be a function of among other things their drug and alcohol use; is that correct?
A. I disagree with the way you phrase the question.

I can't answer it yes or no.

Q. How can you answer it?

A. If you are talking about, let's say, exposure related liver damage, I would assume that alcoholism would make a subject more vulnerable to the PFOA associated adverse affects, but I don't have any evidence to support that. So this would be a medical assumption and I am not sure this is what you are after.

Q. Among the individual differences would be a function of among other things body weight and body mass index; is that correct?

A. Again, I have the same problem in understanding it. Because body mass and obesity could be an affect of PFOA exposure. And therefore to say that obesity or body weight determines the outcome would be to say that the affect of PFOA exposure is affecting the affect of the PFOA exposure. So that sentence does not make sense.

Q. Please turn for me to Page 59. Line 8 of Doctor Ducatman's deposition transcript.

A. (Witness complies).

Q. Doctor Ducatman was asked this question and gave this answer under oath at his deposition. Question, and those individual differences, would be a function of

among other things their body weight and body mass index or BMI, correct, and his answer was one word. Yes. Do you see that?

A. Yes.

Q. Was Doctor Ducatman wrong?

MR. WHITLOCK: Objection to the form.

A. Was Doctor Ducatman what?

Q. Was Doctor Ducatman wrong?

A. At the time, he was probably correct. And at the time that he wrote his expert report that very recently I published a study that showed exactly what I just told you and Doctor Ducatman may not have been, you know, familiar with that study, and also a second study that was just published as of one or two-weeks ago.

Q. So when did you publish the first study that you just referenced?

A. I don't remember. It may have been May or June of this year.

Q. What was its title?

A. Can I -- I don't remember the title. I can tell you what it refers to. It is a project called Pounds Lost. This was a clinical controlled trial where overweight or obese individuals from Baton Rouge and Boston used different approaches to dieting. All of

them had calorie restriction and were therefore exposed to lose weight. And they did over a six-month period. Then they were followed for another 18 months. And during that time, I mean, they all basically used, lost the same amount of weight in the six months, but afterwards they sort of had different paths and some people essentially kept their weights low and some people increased their weight. Do you want me to go on?

Q. No. I believe your paper was actually published prior to Doctor Ducatman's deposition. Okay. Let's just move on.

In terms of the considerable individual differences for exposure, PFOA blood levels, and susceptibilities if any to PFOA and those individual differences would be a function of among other things, one's general state of health as well as other medical conditions, correct?

MR. WHITLOCK: Objection to the form. Compound question. I object to counsel's testimony as to what is considerable and what is not. You can answer if you understood the question.

A. Of course one's -- may I have the wording again?

Q. Why don't we take a look at Doctor Ducatman's deposition transcript. Page 59. Line 13. The question

was, and those individual differences would be a function of among other things their general state of health as well as other medical conditions; is that correct?

A. I would agree with Doctor Ducatman's answer, which is yes.

Q. Those individual differences would be a function among other things their occupational histories; is that correct?

A. Do you mean individual differences in exposure to PFOA?

Q. Yes.

A. There could be some occupational exposure to PFOA. I would agree to that.

Q. In your opinion, among these considerable individual differences and exposures, blood levels, susceptibility to PFOA, if any, is there anything else that those individual differences could be a function of?

MR. WHITLOCK: Objection. Vague.

A. Nothing that I can remember right now. I think we went through a long list already.

Q. Fair enough. Would you agree that one of the hallmarks of science is the requirement of valid and reliable data?

A. That's the way I practice research.

Q. In your opinion, is it important to assess all of the available data relevant to the question at hand before arriving at a conclusion?

MR. WHITLOCK: Objection to the form. Vague.

A. When you say relevant, it has to be considered relevant from a research point of view. So in that sense, I agree with you.

Q. Do you agree no study can be assessed in isolation and that all evidence based literature is needed to form a valid and reliable opinion in science and medicine?

A. With the exception of experimental astronomy where we cannot repeat the experiment, climate change. Within the narrow sense of my work at least, I would not rely on a single study in isolation unless it is supported directly or indirectly by other evidence.

Q. So you would agree that all available papers should be considered in a scientific deliberation and that the selective consideration of the literature is not a scientific procedure in the area in which you worked; is that correct?

A. I don't like it when you say available. Because

you may have a different understanding of that than I do because I rely on the strength of evidence. So I am looking at the strongest evidence that is available and I would not consider the studies that don't have much to do with the conclusions that I need to consider.

Q. Do you agree that selective cherry picking of data is inconsistent with a valid and reliable scientific methodology?

A. That I would do. That's what I have seen expert witnesses do.

Q. I am not sure I got an answer to that. Do you agree that selective cherry picking of data is inconsistent with a valid and reliable and scientific methodology?

A. Right. I agree in that sense that perhaps some experts like the defense experts have their own way of selecting the evidence and they may not call it cherry picking, but I do if it is really picking the evidence in accordance with the conclusions that they apparently want.

Q. In your opinion, is it scientifically valid to use one hypothesis to prove another hypothesis?

MR. WHITLOCK: Objection to the form. Vague. You can answer.

A. A hypothesis is just what it is. It is a hypothesis. So if it is still a hypothesis, you cannot rely on that when you want to draw conclusions about another.

Q. In scientific writings, what does the word suggests or suggestive of mean?

MR. WHITLOCK: Objection to the form.

A. That's one of the words that we try to avoid. It says that the, there is a hypothesis here, and it may be valid, but if it is valid, you would say, shows, but suggests sort of a vaguer term that doesn't indicate that the hypothesis is proven.

Q. Would you agree that it is important to continually re-assess one's conclusions as new information becomes available?

A. I would agree.

Q. Have you ever been wrong or mistaken about something that you believed to be true?

A. I was wrong when I participated in the expert panel on PFOS and PFOA on behalf of the European Food Safety Authority under the European Union because at the time, this was I believe in 2008, and I cite that report in my expert opinion, we considered that PFOS and PFOA were much less toxic than we understand they are now.

So I must admit I was wrong simply because we didn't have enough evidence at that time. So I had to take that back.

Q. When you say you had to take that back -- let me ask you a general question.

When you recognized that you had been wrong or mistaken about something that you had believed to be true, what do you then do in those circumstances as a general proposition?

A. What I do is to publish as soon as I can my new findings that cast out upon the previous conclusions, but I cannot publish a new opinion on behalf of EFSAT for example. I think they need to be redone and they are redoing it now.

Q. What is the ad hominem fallacy?

A. Ad hominem -- I think it is a legal term. All I can say is my understanding of it. It is like you blame, let's say, an expert rather than blaming the opinion expressed by that expert.

Q. Is it fair to say the ad hominem fallacy is a strategy whereby one attacks the character, motive or other attribute of the person making a logical statement rather than attacking the substance of the statement itself?

MR. WHITLOCK: Objection. Asked and answered. Objection to the form. Compound question. Confusing.

A. I think your explanation is just an extension of what I gave before. So I would agree.

Q. In your opinion, is the use of the ad hominem fallacy a proper part of scientific criticism?

A. Let me answer it this way, because you have to take into account that as some people may have vested interests and they, if they are being confronted with that, they may themselves call that an ad hominem attack where it really is a matter of conflicts of interest. So in that sense, I would say conflicts of interest are very important in our judgment of evidence and writings.

Q. What factors do you believe give rise to a conflict of interest?

A. I think it is preferable to refer to the definitions given like the International Group of Medical Journal Editors. Because they refer to, well, I don't know this by heart, but as a journal editor, I am familiar with these issues, and we prefer to refer to transparency so that readers can understand the situation in which the author has expressed an opinion. It may be that most readers will not see that as a

conflict and other readers who may understand that this was part of, let's say, a legal case that they will say, there is a conflict of interest here.

Q. In your opinion, does funding give rise to a conflict of interest?

A. It depends on the source of the funding.

Q. In your opinion, is funding the only thing that can give rise to a conflict of interest?

A. No. Again, I refer to the International Committee of Medical Journal Editors. They considered for example stock ownership and they considered the spouse's stock ownership and issues like that.

Q. In your opinion, can an agenda to accomplish a social or health care purpose no matter how well intentioned give rise to a conflict of interest?

A. I can't answer that in general. Because it depends on what you are really referring to here.

Q. Is it fair to say ambition to find something original is common among scientists?

A. I would say so.

Q. Would you agree that there is a certain amount of pride that comes from being the first scientific investigator to report a new finding?

A. I would agree.

Q. On Page 19 of your report, in the last paragraph, you assert that several of Doctor Mandel's PFAS related articles were published in a journal known favor to submissions from industry doctors, and on Page 21 in the carryover paragraph, you assert that some Dr. Calabrese's articles are published in journals well known to favor submissions from industry toxicologists; is that correct?

A. That's what I say here.

Q. Based simply on where Doctors Mandel and Calabrese have published their work, is it your opinion that the findings that they reported in those articles are invalid?

A. No. I have published in those very same journals myself.

Q. Does where they publish their work make their opinions in this case any less accurate or valid?

A. Not taken alone.

Q. Isn't it true that more than 80 percent of drugs discovered and developed over the past forty years have come from industry?

A. That I don't know.

Q. On Page 21 of your report, you state that according to the Web of Science, Professor Calabrese's

work has been cited a several thousand times over the years. However, about twenty percent of the total are a auto citation. That is, where the author cites his own work.

MR. WHITLOCK: Where are you on Page 21?

A. It is the last full paragraph on Page 21. According.

Q. Why if at all should the percentage of so called auto citations influence your interpretation of another expert report in this case?

A. It does not by itself. But it is part of the whole picture that when Professor Calabrese has achieved as many as several thousand citations, then it is unusual that twenty percent of them come from his own writings.

Q. How did you come up with the twenty percent figure?

A. If you know the website called Web of Science, what you do is to ask for the statistics of the citations and the Web of Science will provide you with a number of citations in the journals that they cover. So he may have many more quotations or citations like that and they also provide the auto citations by number. So that's very easy to do.

Q. Do you ever cite your prior papers in your subsequently published papers?

A. I do.

Q. Have you ever calculated or checked the Web of Science to determine what the percentage of auto citation is for your own work?

A. I have not checked the exact number.

Q. On Page 22 of your report, in the second paragraph, you cite the Ruden and Hanson paper that addresses Doctor Guzelian and related positions about evidence based causation toxicology; is that correct?

A. That's correct.

Q. Were you aware that Doctor Guzelian published the response to Rudin and Hanson?

A. I don't remember it by heart, but I think I saw it.

MR. WOLFF: Mark this as Exhibit Number 3.

(Clear Path Towards an Evidence Based Toxicology EBT was marked as Exhibit Number 3 for identification)

Q. Exhibit 3 is a paper entitled Clear Path Towards an Evidence Based Toxicology EBT. Is this the paper that you were referring to?

A. Yes.

Q. Did you read this paper, Exhibit 3?
A. It is a while ago. I believe I read it.
Q. You do not discuss or cite Doctor Guzelian's response to Rudin and Hanson anywhere in your report, do you?
A. I don't remember.
Q. It is not listed on your reference list, is it?
A. If you say it is not, then I am willing to believe that.
Q. Why don't you cite or discuss Doctor Guzelian's response in your report?
A. As I remember when I wrote this, there was not anything new in this response than in his original presentation of what he calls evidence based toxicology.
Q. Please turn to Page 36 of Doctor Ducatman's deposition transcript beginning at Line 18.
A. 36?
Q. 36. Line 18.
A. Okay.
Q. Is it fair to say all human beings are exposed to thousands of chemicals, that human beings have many infectious diseases, and that human beings are a complex product of their environment and their genetics?
MR. WHITLOCK: Is that a question for Doctor

Grandjean?

MR. WOLFF: Yes, that is.

MR. WHITLOCK: Or are you reading it and asked him if he read it correctly?

MR. WOLFF: It is a question for the witness.

A. That sounds reasonable to me.

Q. When considering the effects of exposure to a chemical, shouldn't one consider what is known, what is not known, and what the external risk factors are for the individual?

A. I would not phrase it that way. It sounds reasonable.

Q. Have you spoken to any of the individual plaintiffs?

MR. WHITLOCK: Objection.

A. From this case?

Q. From this case.

A. No.

Q. Have you examined any of the individual plaintiffs in this case?

MR. WHITLOCK: Objection. Outside of the scope.

A. No. No, I have not.

Q. Have you reviewed any medical records for the individual plaintiffs in this case?

MR. WHITLOCK: Same objection.

A. It was not in my -- I was not asked to do that.

Q. Have you reviewed any of the deposition transcripts for any of the individual plaintiffs who have been deposed in this case?

MR. WHITLOCK: Same objection.

A. I don't remember so. If it is not listed in my report, it is because I have not.

Q. As of the time that you issued your report, did you have any plaintiff specific information as to the average daily consumption of water by the individual plaintiffs?

MR. WHITLOCK: Same objection.

A. Not that I remember. If I didn't state that in my report, it is because I didn't have that information.

Q. As of the time that you issued your report, did you have any plaintiff specific information as to the percentages of tap water versus bottled water versus activated charcoal filtered water typically consumed by the individual plaintiffs?

A. The answer is the same.

Q. Is the answer no?

A. The answer is that I didn't have that information as long as I remember.

MR. WHITLOCK: Objection to the question. Outside of the scope.

Q. Do you know whether the water consumption practice and patterns of the proposed class representatives are typical of the water consumption practices and patterns of absent class members?

A. Again, that was not in my charge and I don't remember having seen any such information.

Q. Were you aware that the individual named plaintiffs in this matter have submitted sworn answers to interrogatories?

MR. WHITLOCK: Objection. Calls for legal conclusion. Outside of the scope.

A. Again, I am not aware of this.

Q. So you have not reviewed any of the interrogatory answers from any of the individual named plaintiffs; is that correct?

MR. WHITLOCK: Objection. Asked and answered.

A. Because it was not in my charge. So I have not reviewed that information.

Q. Starting at the bottom of Page 9 of your report,

you refer to a study of PFOA concentrations in the blood of Bennington residents, and you note that the geometric mean was ten micrograms per liters; is that correct?

A. That's correct.

Q. At the end of that paragraph, you refer to the named plaintiffs and state that their blood serum values are very high and range between 24.8 micrograms per liter and 305.1 micrograms per liter?

A. Yes.

Q. At the low end, among the individual plaintiffs, the 24.8 micrograms per liter is two-and-a-half times greater than the ten microgram average among the Bennington residents that were tested; is that correct?

A. I think that is correct, yes.

Q. At the high end among the individual plaintiffs, the 305.1 micrograms per liter is thirty times greater than the ten microgram per liter average among the Bennington residents that were tested; is that correct?

A. Right. But it is also in the upper five percentile. So it is within the range.

Q. Are you an immunologist?

A. If I'm an immunologist? No. I am not. I have not claimed to be so.

Q. Are you an internist?

A. No.
Q. Are you a hepatologist?
A. No.
Q. Are you board certified in any particular field of medicine?
A. I decided not to go for that.
Q. Do you hold any special certifications in epidemiology?
A. There was not such a thing when I started working in this field.
Q. The answer is that you do not?
A. I don't know. I do not. I have taught people so they could get those certifications.
Q. Do you hold any special certifications in toxicology?
A. No.
Q. Are you licensed to practice medicine anywhere in the United States?
A. No.
Q. Have you ever been -- strike that.
Have you ever been licensed to practice medicine in the United States?
A. I have not.
Q. Are you currently licensed to practice medicine?

A. No.

Q. Following your graduation from medical school in January of 1974, did you ever complete a medical residency?

A. I got a research position right away. So I skipped it, but I did work as a physician at Mount Sinai Hospital in New York City.

Q. Do you regularly see patients in a clinical setting?

A. Only in connection with immunological studies. They are not patients. They are members of populations.

Q. Do you physically lay hands on those patients?

A. No.

Q. How often do you see patients in a clinical setting if at all?

A. That is hard to answer because I worked at Mount Sinai Hospital for two years. Most of what I did was in connection with population studies.

Q. When was it that you worked at Mount Sinai?

A. That was in 1978 and 1979.

Q. So a while ago?

A. A while ago.

Q. As part of your current practice, do you diagnose disease in patients?

A. No.
Q. As part of your current practice, do you prescribe medication or other therapies to patients?
A. I do. I can. And I do. Only to my immediate family.
Q. How long has it been since you regularly saw patients in a clinical setting?
A. Again, I have to say I see subjects who are members of populations that we study. I do not deal with individual patients.
Q. How long has it been since you regularly saw individual patients in a clinical setting?
A. I would say we have to go, formally we have to go back to 1979.
Q. When was the last time that you prescribed medication to or other therapies to patients who were not members of your immediate family?
A. I try not to because I refer colleagues or friends to their own physician; but I do occasionally if it is a matter of urgency, I do prescribe what I believe is medically responsible. I don't remember if that is a year ago, but something in that order of magnitude.
Q. How long has it been since you regularly diagnosed disease in particular patients in a clinical

setting?

A. Well, I have to go back to Mount Sinai again.

Q. 1978, 1979?

A. 1979. Yes.

Q. How long has it been since you regularly prescribed medications or other therapies to individual patients in a clinical setting?

A. I think, when you ask a question that way, I should say I did make a decision to leave clinical practice very early.

Q. How long has it been since you last prescribed medicines or other therapies to individual patients in a clinical setting, are we talking again the late 1970s?

A. Yes. Not counting my friends and family.

Q. How long has it been since you last used a stethoscope to listen to the hearts and lungs of an individual patient in a clinical setting?

A. That was a long time ago because I got some hearing problems when I was in my forties.

Q. So --

A. I would have to go back to 1979.

Q. So you are an epidemiologist?

A. I am. I am.

Q. Epidemiology deals with the study of diseases in

populations and factors associated with it, correct?

A. Correct.

Q. And clinical medicine is intended for individuals; true?

A. That's the way I look at it.

Q. Have you ever designed a population based medical monitoring program?

A. I have. Because I was part, I was the toxicology expert for the, I am thinking of the translation. It is like the Board of Public Health within the Minister of Health in Denmark. I was in that position for over thirty years, I believe, and helped the Board of Health on matters like this. I am not sure if we used the term medical monitoring, because it always depends on what is available already; but these were discussions that we would have in the Board of Public Health. And also before that time when I was chief of occupational medicine for the labor department, I helped decide on the new regulation on lead exposure, occupational lead exposure and that would be required of workers with such exposure in regards to medical services, blood testing that should be available to them.

Q. When you said that the medical monitoring depends on what is available already, what did you mean by

that?

A. Well, the setting in Denmark is different from here, because it is a national health service. What one would request is something that's not available already.

Q. For clinical laboratory tests, their sensitivity, their specificity, positive predictive value in a population at issue for diagnosing a particular health endpoint are expressed numerically; is that correct?

A. That's correct.

Q. In terms of clinical laboratory tests, you do not use the terms sensitivity or specificity in the text of your report, do you?

MR. WHITLOCK: Objection. Outside of the scope.

A. I don't remember if I did. This is not what I was asked to do.

Q. In terms of clinical laboratory tests, you only use the term predictive value once in the text of your report. Why is that?

MR. WHITLOCK: Same objection.

A. It was not my primary charge. Maybe I shouldn't have even mentioned this. Yes.

Q. As to the various health endpoints, do you know the sensitivity, specificity and positive predictive

value in the population at issue for each of the clinical laboratory tests that Doctor Ducatman suggests?

MR. WHITLOCK: Objection. Form. Compound question. Outside of the scope.

A. If I needed to, I could look it up. This was not what I was asked to do.

Q. As we sit here today, do you know the sensitivity, specificity or positive predictive value in the population for any of the clinical laboratory tests that Doctor Ducatman suggests?

MR. WHITLOCK: Objection to the form. Vague. Outside of the scope.

A. I don't know it by heart.

Q. Gamma-glutamyl trans peptidase, if this test is positive, what specific disease if any is this test for?

MR. WHITLOCK: Objection. Outside of the scope.

A. I'm not sure I understand that word.

Q. Gamma-glutamyl trans peptidase?

A. Gamma what? I think that is an error. I don't know. I am not sure that exists. If you mean the transfer, it is a test that is routinely used for liver function. Pepsin is a stomach enzyme. These are my abbreviations. Here it is.

Q. I see. On Page 72 of your report, you refer to GGT?

A. Yes.

Q. So if the GGT test is positive, what specific disease if any is that test for?

A. I am not prepared to answer that question because it is outside of my report; but it is usually and has been used here as an indicator of abnormal, if it is elevated, abnormal liver function.

Q. Do you know if it is a test of any particular liver disease?

A. I don't know that by heart.

Q. Do you know the sensitivity and specificity of the GGT test for diagnosing liver disease?

MR. WHITLOCK: Objection. Outside of the scope.

A. Again, it is not something that I know by heart.

Q. Have you ever looked it up?

A. That's a few years ago. This is part of everyone's medical education.

Q. So when you say a few years ago, are we talking the 1970s?

A. No. I checked those matters on occasion because medical knowledge is of course expanding.

Q. Do you know the positive predictive value of the GGT test in the Bennington population?

MR. WHITLOCK: Objection. Outside of the scope.

A. My answer is the same. I don't have any information available on that.

Q. Do you know whether blood testing for GGT levels is among the care that would be provided to someone that sees a doctor regularly?

MR. WHITLOCK: Objection. Outside of the scope. Calls for speculation.

A. No. I don't have that information available.

Q. Would you find it unusual if some of the plaintiffs in this matter have had their GGT levels routinely checked as part of receiving regular care from a physician?

A. I don't have any knowledge available to allow me to answer that question.

MR. WOLFF: Off the record for a moment.

THE VIDEOGRAPHER: The time is 10:10. We are off the record.

(Discussion off the record)

THE VIDEOGRAPHER: Back on the record. The time is 10:11 a.m.

MR. WHITLOCK: Bert, I was going to make the proffer that the plaintiffs will stipulate that we are not offering Doctor Grandjean as an expert in medical monitoring in an attempt to hopefully shortcut some of these questions that we were just going through.

MR. WOLFF: I appreciate that. Let's take a break.

THE VIDEOGRAPHER: The time is 10:12. We are going off the record.

(Whereupon a break was taken)

THE VIDEOGRAPHER: We are back on the record. The time is 10:22 a.m.

Q. Throughout your report, for example, on Pages 16, 23, 26, and 70, you level a criticism at the defense experts for purportedly ignoring a number of prospective studies, don't you?

A. I believe that is what I say here.

Q. While your report otherwise contains many end noted citations and brackets with numbers, when you make those assertions, you do not cite, list, or end note the studies that the defense experts purportedly ignored, do you?

A. I thought I did. If that is something you are missing, it should be very easy to see. They don't cite

very many reports either of them.

Q. So what perspective studies do you contend the defense experts ignored?

MR. WHITLOCK: Objection. Vague. Ambiguous. Point to a specific place in his report if you are going to ask him questions about that.

Q. Page 16. You say a substantial number of perspective studies have emerged. Although this has not been acknowledged by the defense experts. What studies are those?

A. When I say that there is a substantial number, it is very clear, because I think that I have listed many studies in my report here, and I simply have the impression that those reports are superficial, and I don't think that I should be asked to remember those or to cite those numerous studies. It is easy to check if you want.

Q. Please, give me the first ten studies that you would include in that list by first named author and year of publication?

A. I don't think that is a reasonable request. I am sitting here with my report and without the references.

Q. Your references are attached to your report, are they not?

A. I don't know what you mean by that question.

Q. What I mean by the question, if we take a look at your report, which has been marked as Exhibit Number 1, Exhibit D contains your cited publications in the report ranging from Numbers 1 through 277. So among those 277 citations, please, list for me up to ten that you contend are prospective studies that the defense experts ignored.

MR. WHITLOCK: Objection. It implies these studies are included in Exhibit D to Doctor Grandjean's report.

A. I can not do that on the spot. If it is very important to you, I can do so later this month and I can report the result to counsel.

Q. On Page 1 of your report, you state that you are an adjunct professor at the Harvard School of Public Health?

A. That's correct.

Q. Being an adjunct professor is not a tenured track position; is it?

A. I was offered a tenured, full professor position at Harvard that would pay my salary and pension; but I decided not to up for that because I wanted to keep my laboratory and my cohort studies overseas.

Q. Being an adjunct professor is not -- strike that.

Being an adjunct professor is not a tenured track position, is it?

A. I have a ten -- not tenured track. I have a full tenured lifetime position as full professor. I decided to be adjunct at Harvard so that I would be able to work there and do my research there at my own pleasure.

Q. Being an adjunct professor is not a tenured track position, is it?

MR. WHITLOCK: Objection. Vague. Asked and answered twice.

A. Already said that. I chose, I was given the option, and I chose to become an adjunct professor.

Q. As adjunct faculty, your primary affiliation is with another institution, and in this instance, in Denmark, true?

A. I was named professor when I was 32 and became chairman and also have been vice dean at my medical school overseas.

Q. In Denmark?

A. In Denmark.

Q. And your title at Harvard is based on your title at your institution in Denmark; is that correct?

MR. WHITLOCK: Objection to the form.

A. Well, if you want the whole story. It went all the way to the chancellor's office to figure out if they could name me a full professor at Harvard because that is what they wanted. Because I preferred to maintain at least a part-time access to my own laboratory and cohort studies, Harvard said that they would have to make me an adjunct professor which I accepted.

Q. So you are not engaged full-time at Harvard, are you?

A. No.

Q. You do not have the same rights and privileges as the non-adjunct faculty at Harvard, do you?

A. I think I do.

Q. As an adjunct faculty member, you do not have the privilege of voting in schoolwide faculty meetings, do you?

A. I think I do; but I rarely can attend. It is not relevant to me.

Q. Do you have your own dedicated administrative staff at Harvard or do you share the administrative staff with others?

A. I have my own staff.

Q. Do you have your own dedicated office at Harvard or do you share office space with others?

A. This gentleman was in my office yesterday. I do have my own office.
Q. Do you receive a salary from Harvard?
A. I do.
Q. Let's talk a little bit about scientific methodology.
A. Okay.
Q. Can we agree the essence of science is the scientific method?
A. If you say so.
Q. Would you say so?
A. It depends on what you understand by method. I sort of hesitate this is always true.
Q. Can we agree the essence of the scientific method is hypothesis and refutation?
A. Hypothesis refutation?
Q. Yes.
A. I don't get that.
Q. The scientific method at its essence proposes a hypothesis and then attempts to refute that hypothesis?
A. In agreement with the -- yes.
Q. At the bottom of Page 22 of your report, you state that critique may be considered appropriate for highly respected experts and may appear in

accordance with their high methodological standards and un-relenting scepticism to the work by younger colleagues. However a narrow focus on scientific methodology is often coupled with blindness to environmental degradation and social injustices. Not surprisingly, the strategy of criticizing research methodologies has been vigorously explored by vested interests often with the purpose of manufacturing doubt. Although it may serve an educational purpose to demand an almost unrealistic quality level of research, it usually aims blocking prudent risk management while paving the way for potentially toxic chemicals to be released in the environment. Close quote.

Do you see that?

A. I see that.

Q. What did you mean by that statement?

A. I think exactly what I quoted here. If you have any questions about that statement, I am happy to answer that.

Q. In your view, is it inappropriate to assess whether an opinion being advanced by scientists is the product of reliable principles and methods?

A. I need that again.

Q. Sure.

In your view, is it inappropriate to assess whether an opinion being advanced by a scientist is the product of reliable principles and methods?

A. It relies on -- it sounds reasonable.

MR. WHITLOCK: Objection. The question was vague.

Q. Is it appropriate to assess whether an opinion being advanced by a scientist is the product of reliable principles and methods, is that appropriate or inappropriate?

A. Am I using the methods or is it another scientist using appropriate methods? I don't quite understand.

Q. Let's say a third party wants to come in and take a look at your opinions in this case. Is it appropriate for a third party to assess whether the opinions that you are advancing in this case are the product of reliable principles and methods?

A. Yes. That's reasonable.

Q. In your view, is it appropriate to assess whether one has reasonably applied principles and methods to the facts of the case?

A. I agree with that, too.

Q. Is it your position that in assessing potential environment health-related disturbances, having high

methodological standards or a narrow focus on scientific methodology or an almost unrealistic quality of research is inimical to prudent risk management, and doing so is tantamount for paving the way for potentially toxic chemicals to be released in the environment?

A. I think you are citing from what I just said. I stand by that of course.

Q. Is that your opinion?

A. That is my opinion.

Q. In making the assessments described in your report, did you in any way lessen your focus on scientific methodology in order to ensure a precautionary risk assessment approach?

MR. WHITLOCK: Objection to the form.

A. I did not use different principles.

Q. Was David Michael's article in Scientific American a plea to use less than the highest quality science possible in environmental risk management?

A. That would be a misunderstanding. I also -- he wrote a book about this. In addition to the Scientific American article.

Q. In your assessment of the defense expert reports, do you contend that any of those experts did their work

with the intent of producing doubt?

A. It is something that appears to be so. At least in my judgment.

Q. You are familiar with Karl Popper's scientific theory of conjecture and refutation?

A. Yes.

Q. Do you disagree that refutation or theory falsification should be used sparingly in matters of environmental health?

A. What I agree is that we should use, you know, the strongest possible scientific methods to the extent possible.

Q. So you say to the extent possible. What does that mean?

A. It means, for example, that we can't achieve the highest level of scientific proof by dispensing PFOA to experimental subjects. That is unethical and illegal.

Q. On Page 14 of your report in the first full paragraph, you state that observational studies will rarely if ever provide a one-hundred percent proof. Do you see that?

A. I see that, yes.

Q. Is there such a concept as 75 percent proof or fifty percent proof?

A. No.
Q. As used in the concept of your report, what is your definition of proof?
A. We don't use that term regularly but what we say is that the evidence is convincing or not convincing.
Q. How do you know when something has been proven?
A. As I said before, it is not a term that we would regularly use because you can always raise doubt which means that the perfect proof does not exist.
Q. Let's turn for a moment to words that you regularly use. The word association is not a substitute for the word causation; is it?
A. That's correct.
Q. In fact, Doctor, wouldn't you agree that distinguishing between concepts of causation and association is an important area of scientific discussion?
A. Yes. I would agree so in general terms.
Q. Wouldn't you agree that a number of different types of relationships may exist between an exposure and an outcome and one type may be a spurious association resulting from chance bias or confounding?
A. It is possible.
Q. Would you agree that three general categories of

phenomena can result in an association found in a study to be erroneous, chance, bias and confounding?

A. It is possible.

Q. Does a statistically significant finding in a reliable epidemiological study that looks at the disease risk as a function of exposure necessarily mean that there is a causal relationship?

A. That is correct and I assessed that in my report.

Q. When you say it is correct, are you saying that a statistically significant association means that there is a causal relationship?

A. No. It is an issue that one has to consider whether it exists. It is not a necessary condition.

Q. Would you agree that if bias or confounding affects a study, it can invalidate an association that the study found even if it was a statistically significant association?

A. That is always possible.

Q. Can confounders be both known and unknown?

A. That is correct.

Q. There can be known and unknown sources of bias in an epidemiological study; true?

A. I believe so.

Q. When you read the report of an epidemiological

study, what factors do you consider in order to determine whether the results are supportive of a cause and effect relationship between the exposure and disease?

A. I think you mentioned at least most of the factors that I would look at.

Q. You would agree that the statistical significance test is a test of the data and not of the hypothesis being advanced; is that correct?

A. That's correct.

Q. In a study with a P value of 0.05, if the 95 percent confidence interval includes one, then the data are not statistically significant for that finding, are they?

A. That is a correct interpretation.

Q. Epidemiological studies never establish causation in a particular person, do they?

A. In a single person, no.

Q. What is a cross sectional study?

A. A cross sectional study is one where all the relevant perimeters are obtained at the same time.

Q. So cross sectional studies examine both the exposure of interest and individuals with or without the disease of interest at a single point in time; is that

correct?

A. That's correct.

Q. While cross sectional studies can determine the prevalence of disease, they do not determine the incidence or risk of disease; true?

A. I would not say so in general, because it depends how cross sectional it is if you are just looking at exactly the status today; but, maybe, you know, a time varying of information that you are obtaining cross sectionally. So I can't say that this is always true.

Q. What are the limitations of cross sectional studies?

A. What are verications?

Q. No. What are the limitations of cross section studies?

A. I think you mentioned the time factor. That is certainly important because in a study like this, you would like the exposure to occur before the outcome in order to support, possibly support hypothesis of exposure being hazardous.

Q. In cross sectional studies, because both exposure and disease are determined at the same point in time, such studies do not demonstrate that exposure precedes the disease, do they?

A. Again, I hesitate to say yes, because if we talk about PFOA, we are here and if you mean the exposure in terms of the concentration in the water in the household, I would say the concentration of the water, let's say, it has changed over time. For example, because a filter has been installed. Then it would be wrong to associate the concentration of PFOA in the water exactly today with the health status of that household. On the other hand, if you talk about blood PFOA concentrations, then they are a result of accumulations over time and it is not a typical cross sectional study for that reason. Because the blood concentration reflects what has happened over several years in the past. Therefore, one should not just ignore that information because it has to do with accumulation and then perhaps it may be diseases where the actual status today is also something that has been established over time. So it is not a typical example.

Q. Merely because a person lives in an area for, let's say, one year, that has PFOA in the water system does not tell you how much PFOA is in their blood serum, does it?

A. It tells me that the concentration will be elevated. It does not tell me the exact number if that

is what you are after. It does not tell me the exact number.

Q. And the extent to which the concentration might be elevated would be a function of the amount of PFOA in the water system; is that correct?

A. Right. There is no doubt that it will be elevated.

Q. It is also a function of how much water an individual drinks a day, for example, if an average person, if a person on average drinks one glass a day and another person drinks five glasses a day, their blood serum levels are likely to be different; true?

A. Yes, but they will all be elevated. This is also what the state found.

Q. So, for example, in this case, among the individual named plaintiffs, we have PFOA blood serum values ranging from roughly 28 micrograms per liter all the way up to 305 micrograms per liter; is that correct?

A. That's what we went through before.

Q. The geometric mean in the Bennington area for the people tested was 10.1 micrograms per liter?

A. Give me that number again.

Q. 10.1.

A. I think so, yes.

Q. So there is a wide range of blood serum levels?
A. Yes.
Q. Because cross sectional studies measure exposures and health conditions simultaneously, isn't it fair to say in cross sectional studies, the temporal relationship between an exposure and a disease cannot be inferred?
A. I need to clarify this because, as I said before, if the exposures are a matter of the consumed concentration of PFOA, it does go years back and therefore you cannot just conclude that it is a typical, cross sectional study where everything is measured at the same time and therefore it is not as strong. In this case with PFOA, it is actually stronger than a typical cross sectional study.
Q. Why is that?
A. Well, because I explained before that the concentration of PFOA that you have in your serum today, that is a result of your exposure in recent years. Therefore, it is not just something that you get right today. This is your build up in your body and therefore a longer term than just today.
Q. Is it fair to say that cross sectional studies are rarely useful in identifying toxic agents?

A. I think that is an overestimation for the reasons I just told you. In fact, many cross sectional studies actually underestimate the degree of a hazard simply because the association is more or less dwarfed by noise. Statistical noise and variations. Therefore, they are not as strong as prospective studies of course.

Q. What is risk?

A. Risk is the likelihood of an outcome, a probability if you want.

Q. What's the difference between relative risk and absolute risk?

A. Well, the relative in comparison to some reference population say where the absolute risk is the actual risk that the population of interest has.

Q. What is absolute risk?

A. Well, how should I explain this to you. It is a likelihood of this unwanted outcome.

Q. When considering risk, are dose and the hazard important factors?

MR. WHITLOCK: Objection to the form. Vague. You can answer.

A. Insofar as we are relating that risk to a predictor.

Q. What do you mean by a predictor?

A. A predictor would be a hazard. A suspected hazard.

Q. Biological plausibility, when we say something is biologically plausible, that simply means it makes biological sense; isn't that true?

A. That is a nice way of putting it.

Q. You would agree biologic plausibility is not synonymous with causation, wouldn't you?

A. It is something that you take into regard when you judge whether or not there is causation.

Q. Biological plausibility itself is not sufficient to establish causation; true?

A. One would never do that. It is an irrelevant question. I am sorry.

Q. Does the dose response relationship stand for the proposition that the greater the dose, the greater the likelihood for the effect?

A. That's reasonable, yes.

Q. Is it fair to say that it is not only important to know that a substance is capable qualitatively of causing something, but that it is also important to know the quantitative eligibility of the suspected agent?

A. It is if possible.

Q. And isn't there a classic saying that everything

is toxic and it is just a matter of dose?

A. Yes. That's what Paracelsus said three hundred years ago. That you will see among my references that I have more recently of course adjusted that statement because it is kind of a blessing.

Q. In sufficient doses, table salt can be quite toxic; is that true?

A. If you want to call it toxicity, you can claim that you died from eating an enormous amount of salt. I would not consider that toxicity as such.

Q. Oxygen can be toxic?

A. If you want.

Q. What is a threshold?

A. Well, a threshold is the issue that an exposure in this case below a certain level does not represent a hazard.

Q. You would agree that cigarette smoking can cause lung cancer, correct?

A. I would agree.

Q. You would not consider smoking one cigarette in a lifetime to pose a significant risk of lung cancer, would you?

A. Not one cigarette alone. No.

Q. If someone did develop lung cancer after smoking

one cigarette, would you conclude that there was a significant probability that it was caused by the one cigarette?

A. No, I would not.

Q. Do we agree that a temporal relationship does not establish causation, in other words, just because a condition follows an exposure, it does not necessarily mean that the exposure caused the condition, does it?

A. No. It is like, these issues you could say they are nonreciprocal in that it is important to know that is the case, that the exposure comes before the outcome, but the fact that it does not prove anything.

Q. When one considers the effects to exposure to a chemical, isn't it important to distinguish between that effects have been reported and those which are merely feasible or theoretically possible?

MR. WHITLOCK: Objection to the form.

A. I don't understand feasible. What do you mean?

Q. Okay. Let me modify the question.

When one considers the effects of exposure to a chemical, isn't it important to distinguish between effects that have been reported and those which are merely theoretically possible?

A. That makes sense.

Q. Let's talk a little bit about animal data. Isn't it fair to say that extrapolating data from animal models to humans can be fraught with difficulties?

A. Yes. I would agree.

Q. Isn't it fair to say that attempts to extrapolate data from animals to humans must be done with caution?

A. That's correct.

Q. Isn't it fair to say that among other things there can be interspecies differences in metabolic rates, anatomy, cellular biochemistry and the absorption distribution metabolism and the elimination of chemicals?

A. They can be, yes.?

MR. WHITLOCK: Objection to the form.

Q. Wouldn't you agree the results of an animal study will be influenced in part by which species of animals are used?

A. That's correct.

Q. Wouldn't you agree that even among experimental animal models, different species can exhibit different sensitivities or susceptibilities or reactions to various substances?

A. I agree.

Q. Isn't it fair to say that even different strains

of the same species sometimes experience different reactions to the same substance?

A. I would agree.

Q. Let's talk a little bit about the ATSDR which you referenced a bit in your report.

A. Yes.

Q. A what is the ATSDR?

A. The Agency for Toxic Substance and Disease Registry.

Q. The ATSDR is a part of the Federal Centers for Disease Control and Prevention; is that correct?

A. That's correct. And I am going there tomorrow.

Q. Nice. Where is that?

A. In Georgia. It is in Atlanta.

MR. WOLFF: Mark this as Exhibit Number 4.

(Copy of the August 2015 ATSDR was marked as Exhibit Number 4 for identification)

Q. Exhibit 4 is a copy of the August 2015 ATSDR, CDC tox guide for per perfluoroalkyls. Have you seen this document before?

A. Yes.

Q. Please turn with me to the second page. In the right-hand column in the second bullet point, are you there?

A. Okay.

Q. The CDC states that the primary effects observed in animals include liver toxicity, developmental toxicity, and immune toxicity. There are profound differences in the toxicokinetics and mode of action of perfluoroalkyls between humans and experimental animals. Many of the observed affects in animals result from the ability of PFOA and PFOS to activate perisome proliferator activated receptor alpha. PPAR. Alpha. Humans are much less responsive to PPAR alpha than rodents and thus may not be as susceptible to these types of effects. Do you see that language?

A. I see that.

Q. Do you agree with that statement?

A. No.

Q. Why not?

A. Because this is a statement that was prepared in 2015, and that was from a draft toxicology profile from ATSDR and they updated that in 2018.

Q. Is there an updated tox guide for perfluoroalkyls from ATSDR CDC?

A. I don't remember.

Q. When you say they updated that in 2018, what did they update?

A. They updated the tox profile.
Q. Is what you are referring to a several hundred page document?
A. That's correct.
Q. Let's talk a little bit about regulatory standard setting. Okay?
A. Okay.
Q. In the regulatory context where public safety and health policies are at issue, is an assumption of causation often made as opposed to a clinical setting where the criteria for causation are more stringent?
MR. WHITLOCK: Objection to the form.
A. I don't like the wording of that question because in a clinical setting, you do not need to have foolproof of a causation before you do this appendectomy. So you need to clarify what you mean.
Q. Are there different standards for evaluating causal hypotheses when one deals with matters of regulatory public health policy as opposed to hypothesis testing in experimental science?
MR. WHITLOCK: Objection to the form.
A. I am not sure I can answer that question.
Q. Is it a basic tenet of regulatory public health policy to in effect err on the side of caution?

A. I would say so.

Q. Do you agree that the government may regulate or indeed prohibit the use of a chemical despite the low probability of a causal relationship?

MR. WHITLOCK: Objection to the form. Vague. Ambiguous.

A. I would say you use the word may. So I have to say it is possible, but I am not sure I ever saw that happening.

Q. The manner in which one errs on the side of caution in establishing regulatory public health policy is not something that one has to do in a basic science environment, is it?

A. When you say so. I would think that is not what you want to do because no science is perhaps a major input that the regulatory agencies would want.

Q. Is there any reason to err on the side of caution in a toxic tort suit where regulatory public health policy is not at issue but rather than scientific reliability?

A. I can't answer that question. It sounds to me like it is a legal question.

Q. What is the precautionary principle?

A. There are various definitions of precautionary

principle. One of them is from the United Nations. Another one is from a European commission. There is one from the European Environment Agency, but I can paraphrase if you want.

Q. I know you have written on this subject a bit.

A. I have.

Q. How do you use the word precautionary principle, what's your preferred definition of the term?

A. Right. I have to clarify. I don't use it myself. I can discuss what it may mean in regard to interpreting science. I can enter that discussion.

Q. Okay. Please, have at it.

A. So the precautionary principle essentially says that in the face of uncertainty, one should not allow a highly suspected hazard to continue if there are substantial risks associated with that.

Q. The precautionary principle can be summed up with the axiom that it is better to be safe than to be sorry; is that correct?

A. I think that is a very popular term. I would not express it that way myself. It relates to what you just said.

Q. Though you might not express it that way yourself, that's a fair summary; is that correct?

A. No.
Q. Why not?
A. Because it is too far fetching to say better safe than sorry and it is too loose. I don't know what you mean. What do you mean by saying safe, what do you mean by sorry?
Q. Does the precautionary principle inform the premise for regulatory public health policy?
A. I don't think so.
Q. The precautionary principle does not apply to clinical medicine, does it?
A. The way that the European Environment Agency has fine tuned the definition, it is not in regard to preventive medicine, I mean to clinical medicine, but I would say that when a physician judges that child may have appendicitis and we better operate on that, you could say that is an application of the precautionary principle.
Q. Was your opinion in this case informed by the precautionary principle?
A. No.
Q. On Page 63 of your report, you note that the untested chemical assumption is a common concern in risk assessments; is that correct?

A. That's correct.
Q. What is the untested chemical assumption?
A. The wording comes from the National Research Council in this country. The wording means that it is not logical and responsible to assume that an untested hazard has absolutely no affects. Meaning there is no risk associated with it.
Q. Is your criticism of the untested chemical assumption rooted in the precautionary principle?
A. I think those are two different matters.
Q. The EPA's health advisory for PFOA in drinking water is 70 parts per trillion or PPT?
A. Yes.
Q. The Vermont Department of Health has set its drinking water advisory limit for PFOA at twenty PPT; is that correct?
A. That used to be correct, because now that limit has changed to be the sum of PFOA and other major P facets.
Q. In your opinion, these advisory levels are too high; is that correct?
A. That's correct.
Q. You estimate that the exposure limits should be less than one part per trillion, is that correct?

A. Yes.

Q. You arrived at this proposed exposure limit based on a reported association between PFOA exposure and antibody concentrations?

A. That is the evidence that we put forward.

Q. At the top of Page 6 in your report, you wrote PFOA concentrations in drinking water are known to correlate with the serum concentrations of long term residents in Ohio and West Virginia at an approximate ratio of one to one hundred. Do you see that?

A. I don't see it, but I think this is correctly quoted.

Q. Using that ratio then, one part per trillion of PFOA in drinking water would be equivalent to approximately one hundred parts per trillion of PFOA in the blood serum of an individual consuming that water; is that correct?

MR. WHITLOCK: Objection to the form.

A. I am not sure I follow your calculations. If you talk about the ratio we just talked about, then I would agree with you on average.

Q. Just in terms of sort of leveling the various units of measure, one hundred parts per trillion is equivalent to 0.1 micrograms per liter; is that correct?

A. I think so.

Q. Is it your opinion that medical monitoring would be reasonably necessary for individuals with PFOA exposure greater than 0.1 micrograms per liter?

MR. WHITLOCK: Objection. Outside of the scope.

A. This is not what I stated. I stated it would be appropriate for those with an elevated exposure to PFOA. So this is not in accordance with what I wrote in my report.

Q. Elevated in comparison to what?

A. First of all, in comparison with that subject's background. I think that is what this report is about. Because they have been exposed to contaminated drinking water. I am saying, it is the elevation that follows from that exposure that then leads to the recommendation of medical monitoring.

Q. So over the years, NHANES has reported the geometric means of PFOA in blood serum in Americans that have been tested, and so I think that in your report at Page 10, you make note that the current background levels of PFOA in blood serum that the geometric mean is 2.1 micrograms per liter based on the overall US population 2011 to 2012?

A. That's correct. I see that.
Q. So is it your opinion then that an elevated blood serum level for PFOA is something greater than 2.1 micrograms per liter?
A. No. That is not what I say in my report. I refer to the exposure. This is a population that is exposed to PFOA from Saint-Gobain in their drinking water and that leads to an elevated exposure.
Q. Elevated as compared to what?
A. To compare to not being exposed to that drinking water. That is drinking water without PFOA contamination in it.
Q. So would you consider that if a person's blood serum levels had a contribution of 0.1 micrograms per liter from PFOA coming from the tap water, that that would be an elevation as you used the term?
A. I don't quite get the numbers. What we talk about here is a substantial contamination of the drinking water, and where people that live there, and residents, not just people visiting, but residents have been exposed to this, that leads to an elevation.
Q. What would you be consider to be quantitatively a substantial elevation of PFOA blood serum levels against what it would have been had there not been those

exposures?

A. Right. I mean, you can read my paper from 2013 where we calculate that the safe level should actually be about or below 1 PPT. And I stand by that. So meaning that an elevation, I mean, we talked about a manmade compound that does not serve any purpose in drinking water. That these people unwittingly have been drinking for years. I would say that results in an elevation because that substance is not meant to be there.

Q. I am asking what quantitatively is the elevation in blood serum that you would consider to be a substantial elevation as you used the term?

A. I understand. I have not calculated what that might mean, but I refer to the drinking water concentrations, and I have seen the map of all those wells in the community water and it is very clear that they are substantial elevations and exposure.

Q. Well, since the background blood levels of PFOA in the United States exceeds 0.1 micrograms per liter, is it your opinion that every American should be eligible to receive medical monitoring for that exposure?

A. Okay. That's a misunderstanding.

Q. I am asking a question.
A. No. That is not my opinion.
Q. Okay. That was my question. Is that your opinion?
A. Right. My opinion is that is not the case. This is a very specific case with contaminated drinking water. I know the 2.1 is part of the classification, what do you call it, the class definition. That's part, I understand that.
MR. WOLFF: Off the record for a second.
THE VIDEOGRAPHER: The time is 11:12. Off the record.
(Discussion off the record)
THE VIDEOGRAPHER: We are back on the record. The time is 11:13.
Q. In this case, are there substantial elevations of PFOA in the community water supply or is that limited to just private wells?
A. As I recall the numbers, it is in particular in certain private wells.
Q. While everything is toxic at some dose, the exposure levels calculated in a regulatory risk assessment do not tell you what that dose is, do they?
A. Let me try to rephrase the way I understand it.

That the regulatory guidelines or whatever they are refer to particular water concentrations and they cannot exactly be translated into serum levels or serum concentrations or individual disease risks.

Q. Do you consider yourself an expert on the regulatory and administrative procedures for setting an exposure limit?

A. I would leave that to others to decide. I have certainly advised regulatory agencies on which decisions to reach.

Q. In the context of regulatory risk assessments and the setting of regulatory exposure levels, what are uncertainty factors?

A. Well, uncertainty factors they are applied when translating dose levels that have been applied in animal studies to levels that may be considered virtually safe in humans.

Q. Does the EPA apply uncertainty factors to account for the extrapolation of results in experimental animals to humans, inter individual varability including sensitive subgroups, extrapolation from a lowest observed adverse effect level to a no observed adverse affect level, extrapolation of results from subchronic exposure to chronic exposures and database inadequacies?

A. That is my understand.
Q. A risk assessment may incorporate multiple uncertainty factors to calculate a regulatory exposure level; is that correct?
A. That's my understanding.
Q. In the past, uncertainty factors have been described as safety factors; is that correct?
A. That's correct.
Q. Given the methods, assumptions, and uncertainty factors that are used in regulatory risk assessments, the permissible exposure levels that are calculated are intended to be protective not predictive?
A. No. They are intend to be virtually protective for the exposed population.
Q. Virtually protective?
A. I think we use the word virtually because you can never completely exclude, that some adverse affect may happen.
Q. Given the methodology, assumptions and safety factors that characterize regulatory risk assessments, the permissible exposure levels do not provide predictive information about actual clinical risks for exposures that exceed such levels, do they?
A. No.

Q. And given the methodology assumptions and safety factors that characterize regulatory risk assessments, the permissible exposure levels do not provide predictive information about medical causation for exposures, that exceed such levels, do they?

A. I don't fully agree with you because medical causation is probably part of the risk assessment made by that. I mean depending on the circumstances of the agency. So one is of course trying to define a protective limit or guideline so that medical causation would not be a problem.

Q. On Page 4 and elsewhere throughout your report, you note that your opinion was formed by using a weight of the evidence approach; is that correct?

A. Weight of the evidence, correct.

Q. What is a weight of the evidence approach?

A. This is the approach that is commonly used by regulatory agencies in regard to weighing, using that term, the evidence in regard to whether it is reliable and solid and can be applied to in the risk assessment process.

Q. How if at all does a weight of the evidence approach differ from Global Introspection?

A. From Global?

Q. Introspection?
A. Introspection?
Q. Yes,
A. I don't understand the term in this regard.
Q. At the bottom of Page 4 of your report, you criticize the defense experts for using causality criteria that are not in accordance with weight of the evidence procedures, applied by regulatory agencies and international organizations; is that correct?
A. Correct. That's what I said.
Q. On Page 19 of your report, in the second paragraph, you again criticize the defense expert reports and write, this report's, referring to your report, this report's previous sections described proper evaluation of toxicity risks as used by regulatory agencies and international organizations like the World Health Organization; is that correct?
A. That's correct.
Q. And in sum, on Page 70 of your report in the second numbered paragraph, you state that the defense experts rely on strict criteria for causality rather than an assessment of the weight of the evidence as applied by agencies such as ATSDR, EPA, NTP, EFSA and IARC, is that correct?

A. That's correct.

Q. Does the weight of the evidence assessment you performed in this litigation conform to the approaches of the ATSDR, EPA, NTP, EFSA, and IARC in conducting risk assessments?

A. I believe so.

Q. Is it fair to say that regulatory and advisory bodies, such as IARC, OSHA, and EPA, utilize a weight of the evidence method to assess the carcinogenicity of various substances in human beings and suggest or make prophylactic rules governing human exposure?

A. I believe so.

Q. Is it also fair to say that regulatory and advisory bodies such as ATSDR, EPA, NTP, and EFSA utilize a weight of the evidence method to assess noncarcinogenic endpoints of various substance in human being and suggests or make prophylactic rules governing human exposure?

A. I believe so.

Q. Is it fair to say that this methodology results from the preventive perspective the agencies adopt in order to reduce public exposure to harmful substance?

A. That I'm not sure I can answer.

Q. Why is that?

A. I am a scientist. Is not a regulator.
Q. What is confirmation bias?
A. Confirmation bias?
Q. Yes.
A. Well, I mean, I am not sure I can define it here.
Q. Let me ask you this. Confirmation bias can arise when individuals tend to interpret, focus on, and remember information in a way that conforms to their preconceived notions; is that correct?
MR. WHITLOCK: Objection to the form.
A. I don't think that is a reasonable definition.
Q. Confirmation bias can affect scientific decisionmaking, true?
A. It is a theoretical possibility.
Q. Conclusion driven science is not really science at all, is it?
A. I disagree with that.
Q. Why?
A. Because decisionmaking in science, as I see it, it's a product, it follows from an original examination of the question, and, therefore, it's an integral part of science as you try to draw conclusions.
Q. Conducting science to conform with one's preconceived notions is not really science at all, is

it?

A. If you can call it science. But I would say it's biased if it happens the way you describe it.

Q. Where if at all in your report do you explain how a weight of the evidence approach is implemented?

A. I believe that I have done that in a whole section of my report.

Q. Can you please point me to that?

A. Okay. Methodological aspects, and risk evaluation. It starts on Page 11. You have to take the next section, Section 4, knowledge, you have to consider that as well.

Q. Maybe you can point us to the pages where the discussion appears in your report.

A. Right. 11 and onwards to 15 and perhaps even up to 19.

Q. What objective standards, if any, did you use to assign differing weights to different data sets?

A. Exactly the standards that I have described in my report in that I consider the power, the statistical power of the study, magnitude of the study, whether it is cross sectional or prospective and how potential bias has been adjusted for or avoided and factors like that.

Q. Did you employ a method of numerically weighting

or scoring the pieces of evidence you evaluated?

A. No. I decided not to simply because there are several adverse effects. The numbers of studies does not justify an attempt to use particular numbers.

Q. Was the weighting of the evidence a product of your personal judgment?

A. It is always a matter of a judgment. I would say that it is not my personal judgment, but it is a judgment that would be similar to and I can see that from ATSDR's report and up to its most recent report, that it is, we are judging the weight similarly.

Q. Do you set out in your report how much weight you assign to each piece of evidence?

MR. WHITLOCK: Objection to the form.

A. Yes. I believe so.

Q. Where?

A. In the section where I, well, in general terms in the section that I just talked to you about; but then when we go onto the, my description of (inaudible) at individual end points, from Page 27 and onwards, that whole section 8.

Q. On Page 27 of your report, you write, I have cited the most relevant studies. Do you see that?

A. I see that.

Q. How did you decide what the most relevant studies are?

A. This is a matter of the weight of the study because I -- no matter what the conclusion was, whether it contributed to weighty evidence to the basis of, I mean, the scientific base for making an evaluation of the evidence.

Q. When you say weighty, that as you apply it, in terms of your report, is a qualitative rather than a quantitative assessment; is that correct?

A. It is not quantitative, because as I said I am not assigning a number to it but it is a relative weight.

Q. What types of evidence did you weigh in reaching your opinion?

A. I mainly relied on epidemiology, but I did take toxicology studies into regard without reviewing them in great detail and in particular I relied on the opinions summarized by regulatory agencies.

Q. What differing weights did you apply to epidemiological studies as opposed to toxicological studies?

A. Because exactly what we talked about before. Namely, that could be species differences. I weighed

the epidemiology evidence primarily and used the toxicology information in regard to biological plausibility.

Q. Did you evaluate each piece of evidence on its own to determine the weight to assign it?

A. I did.

Q. Was every piece of evidence that you weighed of the same quality?

A. No.

Q. Did the studies you considered vary in quality?

A. Yes.

Q. Did they vary in their limitations?

A. They did.

Q. Is it fair to say that a study's statistical method is important for determining the internal and external validity of that study's findings?

A. That is part of it.

Q. Did every study that you weighed use the same statistical method?

MR. WHITLOCK: Objection to form.

A. I mean, you have to use different statistical methods, but, of course, it is all a matter of probability testing, and you could say that is the same methodology but certainly there are specific methods

that vary from study to study.

Q. Did you evaluate the statistical method used by each study before including it in your analysis?

A. I did.

Q. What was your method for weighting studies according to the statistical method that they used?

A. I don't understand the question.

Q. If you applied different weights to each study, according to the statistical methods, how did you determine what weight to apply?

A. It was not the only aspect. Clearly, if a faulty statistical method was used for the case, I would take that into consideration; but your question is so general it is hard to answer it.

Q. Do you agree -- strike that.

Is it your position that in order to be convinced there is an actual hazard, one needs to have statistical significance?

A. In most cases I would agree. I would also emphasize that even if something is slightly different from traditional statistical significance, we still need to consider it as part of the database.

Q. Did you evaluate the statistical significance of each study before including it in your analysis?

A. What do you mean evaluate the statistical significance, do you mean the methodology or the methods in general, what do you mean?

Q. I mean the findings. Whether the findings were statistically significant.

A. It is part of my evaluation as I said before.

MR. WHITLOCK: Objection. Asked and answered.

Q. If a study reported no significant association between PFOA exposure and an endpoint while another study reported a significant association, would you weight both studies equally?

A. No.

Q. Why not?

A. If there is more to take into account, for example, the size of the study so you can't put that -- that is a misleading question. I am sorry.

Q. What was your method for weighing studies according to whether they reported statistically significant associations?

A. It is part of the weight. As I said to you before, this is qualitative. It is on a relative scale. So it is part of the evaluation.

Q. Relative risk is one measure of the strength for

a statistical association; correct?

A. Run that by me again.

Q. Relative risk is one measure of the strength for a statistical association; true?

A. I would not say it that way, because clearly you can have statistical significance with a small relative risk if the study is large; but you may have a large relative risk that is not significant if the study is small.

Q. Did you evaluate the relative risk or other measures of statistical strength when assigning weight to the evidence that you considered?

MR. WHITLOCK: Objection to the form.

A. Again, you are trying to put me down on the size of a relative risk. That is part of it. It is statistical significance or rather the P value and it is a magnitude of the relative risk. It all is part of that evaluation.

Q. How did you decide how much weight to assign?

A. I think we have been through that because it has to do with the methodology used, the time span covered, the size of the population, the methods used. Also, the methods for ascertaining the outcome. So it is a whole range of parameters. You cannot put the question down

to that simple matter.

Q. As I understand it, you took a look at those factors and perhaps others and then sort of considered them, and I am not saying this pejoratively, but cogitated on them for a while and then came up with a qualitative assessment of how much weight to apply?

A. I would not express it that way. I think you understand it. I would say I agree.

Q. Did you consider evidence that reported findings that were inconsistent with your opinion?

A. I did.

Q. How did you weight those studies?

A. The same way all studies were weighted the same way.

Q. But they did not receive equal weight?

A. No.

Q. What factors did you decide to apply greater weight to one study than another study when those studies had findings that were inconsistent with your opinions?

MR. WHITLOCK: Objection. Asked and answered.

A. Let me just say. When we talk about opinion here, it is not a matter of personal opinion, but it's

an opinion, a conclusion as expressed in this report. So it is a conclusion rather than, so the word opinion is not misunderstood. Obviously, one would weigh those studies equally no matter what the conclusions by the authors, but I consider that in light of whatever else is available in that field, the knowledge, the evidence is available and then I would weigh the different contributions and then emphasize those that were the strongest.

Q. Again, I don't mean this to be pejorative, but the only way to, for somebody to audit independently how you weighed the evidence would be to get inside your head, true?

A. That is not true, because the way that I have evaluated the evidence here is very similar to like we talked about before, what IARC has done, WHO, EFSA, EPA ATSDR, so, I am very similar in my opinion. So you could say the audit is what groups of colleagues have decided when working, and I have worked with those agencies myself in the past, so it is a matter of, you could say, consensus.

Q. On Page 14 of your report, in the middle of the paragraph below Table 1, you write that it is always possible for someone critical of the weight of the

evidence to raise some type of doubt seeking to require additional or improved data before a conclusion can be drawn. Do you see that?

A. Yes.

Q. Isn't it also possible for someone to use weight of the evidence to over endorse an opinion?

A. Theoretically, yes.

Q. On Page 22 of your report, you write, that Doctor Guzelian refers to the criteria for use of scientific data in evidence based medicine as applied in regard to proof of therapeutic effects of drugs for beneficial human use and that his mistake is to apply the same criteria in evidence based toxicology regarding proof of harmful effects. Do you see that?

A. Yes.

Q. Why in your opinion is that a mistake?

A. Because the evidence will never be the same. It would not have the same strength. With drugs, you can conduct clinical control trials, which is the highest level of evidence that we can achieve, and we cannot get that level of evidence from looking at a toxic substance like PFOA simply because it is, like we talked about before, it is both unethical and illegal to those people with PFOA, as a known toxic substance. While we can do

that in regard to drugs where we believe there is a beneficial outcome that may be achieved by using that particular drug. That is certainly not the case for PFOA.

Q. Isn't if the method by which one assesses the results of randomized clinical trials, and assesses the results of observational epidemiological studies, essentially the same?

A. They are not the same, because in the clinical control trial, you have a well defined population. You may even have controls, and blinded controls and you know exactly what the dose is, because you are dispensing it yourself. In regard to environmental toxicants, like PFOA, the dosage is something that can be observed afterwards. And that means that we are less certain what the exact dosage was to those subjects. That very population we talked about before.

Q. In your opinion, should the standard for scientific proof of causation be lower to achieve the goal of preventing exposure to chemical substance?

A. My opinion is that we have to achieve the highest level of proof possible. That is my opinion.

Q. The highest level of proof possible for what?

A. For observational studies, in this case with

PFOA, and then we have to judge that prudently in order to reach a respectable decision on what would we allow our fellow beings to be exposed to.

Q. When you say we have to judge that prudently, what do you mean by prudently?

A. Well, exactly what I said. That you will always be able to raise doubt if you want. Some people do that because they may have a vested interest. You still have to reach a decision, I mean, at least that is what the regulatory agencies have, but can be, and they use the word tolerated. Not accepted, but tolerated.

Q. Are you done with your answer?

A. I thought so.

Q. I want to be sure. I don't want to speak over you.

In your opinion, is it appropriate to lower the standard of scientific proof of causation when exposure to a chemical has already occurred and the only question is what effects if any will result from that exposure?

MR. WHITLOCK: Objection to the form.

A. I think it is a very general question that is hard to understand. It depends on the circumstances.

Q. How about in the circumstances of this case?

A. Can you the rephrase the question?
Q. In your opinion, is it appropriate to lower the standard of scientific proof of causation when exposure to a chemical has already occurred and the only question is what affects, if any, will result from that exposure?
A. I think it is a very theoretical question. I don't think that has ever happened. Not in my mind.
MR. WOLFF: Let's go off the record.
THE VIDEOGRAPHER: The time is 11:42. Off the record.
(Whereupon a break was taken)
THE VIDEOGRAPHER: We are back on the record. The time is 11:53.
Q. The plaintiffs in this case limit their allegations to exposure to PFOA; is that correct?
A. That is what I understand.
Q. Your report's discussion of the epidemiological and toxicological evidence concerning exposure to perfluproalkyl substances was not limited to PFOA, was it?
A. It is focused on PFOA. I take into account relevant evidence on the related substances.
Q. For example, your report also refers to PFOS, PFHXS and PFNA; is that correct?

A. Correct.

Q. Do you agree the biological response to chemical substances may be different even though the two chemicals are part of the same chemical class?

A. It could be.

Q. Do you know if that is the situation with respect to the perfluoralkyl substances?

A. This is not known in detail yet. The state of Vermont has decided to look at the total concentration of PFOA and the related substance in regard to the water limit.

Q. Regardless of what the state of Vermont has done as a matter of regulatory public health policy, as a matter of scientific evidence are the biological responses to PFOA identical to those for PFOS, PFHX, HSXS, and PFNA?

A. In some respects, they appear to be.

Q. Are they in all respects?

A. I don't think so; but it is a judgment call because we don't have all of that comparative evidence to be able to draw conclusions.

Q. And in fact on Page 13 of your report, you write that in studies of exposed communities, exposures are usually mixed and it may be difficult to distinguish

between effects attributable to particular PFASs; is that correct?

A. That's correct.

Q. Do you assign different weights to studies that reported associations involving PFAS substances other than PFOA?

A. I believe just about all of them look at both PFOA, PFOS, and some of these studies also looked at others, but those are the two major PFASES.

Q. Among other ailments, people have been experiencing kidney cancer, testicular cancer, bladder cancer, prostate cancer, steatohepatitis, ulcerative colitis, osteoarthritis, gout, pregnancy induced hypertension and asthma for hundreds of years and long before PFOA was ever synthesized; isn't that correct?

A. You are talking about the occurrence. You are not talking about the risk. So this is correct.

Q. On Page 14 of your report in the second paragraph, you write in the present report, while considering the extent of possible biases, my conclusions are stated in terms of assessing elevated exposure to PFOA results in increased risk of injury or illnesses. In many instances, the existing evidence of hazard is much stronger than that, but I understand this

to be applicable legal standard; is that correct?

A. That is what it says here.

Q. You are not a lawyer, are you?

A. No.

Q. What is your understanding of the applicable legal standard?

MR. WHITLOCK: Objection. Calls for a legal conclusion. You may answer.

A. Again, my civil servant understanding is that it is more likely than not that PFOA is associated with the particular outcomes of interest.

Q. What is your understanding of what the applicable legal standard is based on?

A. It is not really relevant here, because, I am, what I am doing is to assess the weight of the evidence based on the information that is available, and then it is up to the court to figure out whether, whatever the legal standard is, whether what I concluded here is appropriate.

Q. Do you know if there is a difference between the legal standard for regulatory rulemaking and the imposition of liability in tort?

A. I would believe there would be.

Q. Do you subscribe to the principle that generally

researchers should be conservative when it comes to assessing causal relationships?

A. I believe that researchers should be honest and apply the weight of the evidence to reach reliable and prudent conclusions.

Q. Is it fair to say that in assessing causation, researchers first look for alternative explanations for the association, such as chance, bias or confounding?

A. By confounding in my view is part of the bias. So, yes. That is part of the assessment of the weight of evidence.

Q. In Doctor Ducatman's report, he states that no one has been declared to have a PFOA associated condition by any formal body. Do you agree with that statement?

A. You are talking about an individual. That is correct, because risk is something that operates on populations. It is a probability.

Q. Would you describe the quality of epidemiological evidence between cigarette smoking and lung cancer to be irrefutable?

A. I would say so.

Q. Based on the published data, what is the relative risk between cigarette smoking and lung cancer?

A. I don't know. I don't remember that by heart.
Q. How would you describe the quality of epidemiological evidence between asbestos exposure and mesothelioma?
A. I would say that it is much stronger.
Q. Based upon the published data, what is the relative risk between asbestos exposure and mesothelioma?
A. How was that different from the question before?
Q. One was asking you to assess the quality and the other is for a relative risk.
A. A relative risk. So an elevated relative risk, are you asking me --
Q. Let me ask you again.
Based upon the published data, what is the relative risk between asbestos and mesothelioma?
A. It is highly elevated and highly significant, but I don't remember the exact magnitude.
Q. When you say highly elevated, can you give me a number and a range, a ballpark range that you're thinking of?
A. No. Not by heart.
Q. Can you compare and contrast the quality of the epidemiological evidence between cigarette smoking and

lung cancer with the body of data on PFOA exposure and kidney cancer?

A. Which way do you want me to do that?

Q. In any way you feel comfortable.

A. Well, with regard to kidney cancer, we have to say that there are much fewer studies. Therefore, the weight of the evidence is less than it is in regard to tobacco smoking and lung cancer.

Q. Compare and contrast the quality of the epidemiological evidence between cigarette smoking and lung cancer with the body of data on PFOA exposure and testicular cancer?

A. My answer is the same. There are much fewer studies. Therefore, the evidence is less solid.

Q. Compare and contrast the quality of the epidemiological evidence between cigarette smoking and lung cancer with the body of data on PFOA exposure and prostate cancer?

A. I would say that the quality of the studies are generally not different. It is just a number of studies that have been conducted.

Q. Compare and contrast the quality of the epidemiological evidence between cigarette smoking and lung cancer with the body of data on PFOA exposure and

steatohepatitis?

A. My answer is the same.

Q. Compare and contrast the quality of epidemiological evidence between cigarette smoking and lung cancer with the body of data on PFOA exposure and osteoarthritis?

A. My answer is the same. Much less evidence on that.

Q. Throughout your report, you refer to the C8 Health Project; is that correct?

A. That's correct.

Q. On Page 18 of your report in the third full paragraph, you state that these data and the conclusions released by the science panel constitute an important basis for the present report. Based on the results from these studies and an evaluation of the literature, the science panel delivered on, quote, probable links, close quote as summarized in the file report from 2012. True?

A. Correct.

Q. At the top of Page 18 of your report, you note that as a result of a settlement with Dupont, the settlement agreement created a science panel of three epidemiologists that was to conduct research in the community to evaluate probable links between PFOA

exposure and human disease; is that correct?

A. That's correct.

Q. Are you aware that by a negotiated agreed upon settlement, Dupont has been funding a C8 Health Project and a medical monitoring program?

A. I don't remember the details; but I think this is correct.

Q. On Page 18 of your report in the third paragraph, you state that based on the results from these studies and an evaluation of the literature, the science panel delivered reports on, quote, probable links, close quote, as summarized in the report from 2012, correct?

A. This is what you read before. That's correct.

Q. Why did you put the phrase probable links in quotation marks?

A. Because that was a wording that was chosen between the experts and the court as far as I remember. Therefore, it had a specific meaning in this connection.

Q. Do you know what that specific meaning was?

A. I don't remember it, but it was something that, the meaning was in order to satisfy both the court and the scientists.

Q. In order to satisfy the scientists in connection with the way that they would normally go about assessing

causal relationships or something else?

A. I think that they assess the causal relationships and the weight of the evidence the same way they would have done otherwise, but the court clarified somehow to them that they should not try to aim for some more or less perfect proof, but that a probable link meaning that there was a clearly greater risk associated with particular outcomes at elevated exposures to these substances. That's my understanding.

Q. Is it the same standards that the epidemiologists would apply in their academic work?

A. It may depend. I can't vouch for all epidemiologists but I can say that in general I would think that we would aim for stronger evidence of causal relationships and just a probable link.

Q. In which scenario, in which context?

A. In the scientific context. We try to achieve the best evidence possible.

Q. Did you put the phrase probable links in quotation marks because it is not a generally accepted standard in epidemiology or clinical medicine?

MR. WHITLOCK: Objection to the form.

A. I don't have any knowledge of that, but the reason that I did it was simply to indicate that it had

a specific meaning. And if I had not put it in quotation marks, people would say, oh, that's just probable links and whatever popular understanding. To indicate that this was a wording that had been agreed upon with the court.

Q. So you are aware that a so called probable link is an agreed to legal standard and not a scientific standard between PFOA exposure and certain health conditions?

A. I think it is both. It was something that was agreed with the three scientists; but of course I was not part of that discussion. So I cannot be sure.

Q. Were you aware that during its review, the C8 Science Panel was repeatedly reminded by the plaintiffs' counsel not to imply typical scientific standards or methods used to determine causation but rather something quite different?

A. I have no insight into this.

MR. WOLFF: Mark this as Exhibit Number 5.

(Letter dated January 22, 2010 was marked as Exhibit Number 5 for identification)

Q. Exhibit Number 5 is a January 22, 2010 letter from the plaintiffs' counsel in the Dupont litigation, Robert Hillot to the C8 Science Panel. Have you

previously seen this letter?

A. I don't think so.

Q. Please turn with me to Page 5. That is the last page. The last paragraph. Were you aware that the plaintiffs' counsel admonished the panel over the distinction between the probable link standard and the inapplicable causation standard that the panel may be more familiar with from its prior academic and scientific work?

A. I can see that.

Q. Were you aware that the plaintiffs' counsel told the science panel to keep that distinction in mind?

A. No. That is not relevant to my opinion.

Q. Please, turn with me to Page 3 in the first full paragraph. Were you aware that the plaintiffs' counsel emphasized to the C8 Science Panel that the distinction between the causation standard and the more lenient, quote, probable link, close quote, standard of proof is of utmost importance in that it is a fundamental distinction in the level and quantity of evidence necessary to make the determination at issue. Were you aware of that?

A. No. I can understand what it says here. I think my understanding that we discussed before is correct.

Q. Please turn with me to Page 4 at the bottom. Were you aware that the C8 Science Panel was instructed by the plaintiffs' counsel that if there just enough evidence to tip the scales towards a finding of any link between PFOA exposure and any human disease even in the slightest degree, then the probable link standard has been met. Were you aware of that?

A. No. Again, it is not relevant to my opinion.

Q. Why do you say it is not relevant to your opinion?

A. Because we are now in 2018. Most of the evidence that was collected by the three scientists has been published in peer review journals.

MR. WOLFF: Mark this as Exhibit Number 6.

(Letter dated August 2, 2015 was marked as Exhibit Number 6 for identification)

Q. Exhibit Number 6 is an August 2, 2005 letter from the plaintiffs' counsel in the Dupont C8 litigation. Larry Winter, to the C8 Science Panel. Have you previously seen this letter?

A. I don't believe so.

Q. Please turn with me to Page 3. The last paragraph. Were you aware that at least as early as August of 2005, the plaintiffs' counsel told the C8

Science Panel members that we recognize that the imposition of a legal standard on scientific analysis presents various problems and we appreciate that the science panel is accustomed to employing proof requirements that are no doubt more stringent than those that govern and control your work here. You have each confirmed that you can follow the unique, quote, probable link, close quote, standard required in this matter regardless of how inconsistent that standard may be with traditional causation analysis. Were you aware that the science panel was told that?

A. I think the general understanding that I described before remains correct. I was not aware of this particular wording, but I don't think this changes anything in what I just said before.

Q. Were you aware that two members of the C8 science panel subsequently testified under oath in 2011 that their, quote, probable link, close quote, reports are not susceptible to scientific peer review and would not be submitted for publication and peer reviewed scientific journals?

MR. WHITLOCK: Objection to form. Lack of foundation.

A. Well, this contrasts to my understanding, is that

most of what this evidence is about in regards to this C8 panel has actually been submitted to peer review journals and published. This is what I am relying on. I am not simply relying on the probable links reports.

Q. Isn't it fair to say the so called probable link findings do not establish causation between PFOA exposure and any adverse health effects in humans?

A. Again, what you are asking me is to repeat myself that I have the understanding of what the probable links are all about, and that it is something that has been negotiated with the court. So I was not part of that. So I have not taken that into consideration. Nor have I considered these letters.

Q. We are done with those letters.

Do you agree that attributing causation to a particular chemical exposure in a specific instance is a challenge when there has been exposure to multiple chemicals?

A. It is always a challenge.

Q. In the real world, everyone is exposed to many chemicals on any given day; is that correct?

A. Yes, but you have to consider the order of magnitude of those exposures.

Q. Those exposures can be multiplied over a

lifetime?

A. I would not use the word multiply, but the number would be of course larger.

Q. What word would you use?

A. I would just say the number would be greater.

Q. Is it fair to say though some groups of people are exposed to different combinations of chemicals in different doses?

A. That's true.

Q. In your report, you cite several of your own research articles reporting associations between PFAS exposure and reduced antibody concentrations, increased body mass, asthma, and shortened duration of breast feeding based on studies in the Faroe Islands?

A. Yes.

Q. The Faroe Islands are a remote archipelago in the North Atlantic, correct?

A. Some people might say they are remote. They are in between Norway and Iceland.

Q. The Faroese diet is traditionally high in whale meat and whale blubber?

A. I would say it used to be.

Q. As of when?

A. There have been advisories issued by the

government or the medical director of the Faroe Islands to limit the populations use of whale meat and whale blubber. I think the first one was issued, I try to recall, 1998.

Q. Not withstanding those advisories, the Faroese diet continues to be characterized by the consumption of whale meat and whale blubber among other things?

A. That is not correct. It is a population that's characterized by marine food. When I say marine, it is primarily fish. That is species of fish also eaten in the US, and shellfish, and comparatively speaking, the average daily intake at the peak was something like fourteen grams of whale meat per day for adults. So I can't say this was a major contribution to the diet.

Q. The marine based diet of the Faroese population exposes them to a number of environmental chemicals; is that correct?

A. Yes.

Q. Those chemicals exposures include methylmercury?

A. Methylmercury occurs in the meat of whales just like in tuna and other similar species eaten in the United States.

Q. The chemical exposures of the marine based diet include PCBs, is that correct?

A. Yes. PCBs occur in the blubber.
Q. The marine based diet confers exposures to DDT; is that correct?
A. DDT often occurs along with PCBs in marine food.
Q. The marine based diet confers exposures to DDE, correct?
A. DDE is a metabolic of DDT.
Q. And the marine based diet confers exposures to trans-nonachlor; is that correct?
A. I don't believe so. And, yes, it is a very small contribution.
MR. WOLFF: Mark this as Exhibit Number 7.
(Chapter on Human Health in the Faroe Islands was marked as Exhibit Number 7 for identification)
Q. Exhibit Number 7 is a chapter on human health in the Faroe Islands from a national report available from the ministry of environment and food of Denmark. Have you seen this publication before?
A. I'm sorry. I can't remember. I must have seen it.
Q. Help me with the pronunciation, the first named authority? Doctor Weihe, the lead author of this chapter is someone that you have frequently collaborated with in

your studies of the Faroese; is that correct?

A. That's correct.

Q. On Page 177, in the last paragraph, the investigators write that the amount of whale meat consumed during pregnancy is the best predictor for maternal methylmercury exposure; is that correct?

A. Yes.

Q. And continuing, they write that any form of blubber, dried or fresh, seems to be the major source not only for PCB but also for DDT, DDE, and trans-nonachlor.

A. All right.

Q. Those last three chemicals had applications as pesticides; is that correct?

A. DDT did. DDE is a metabolite so it wasn't used by itself. But it's a metabolic tran-nonachlor was used as I understand.

Q. As a pesticide?

A. Yes. It is all history.

Q. The report also states that the Faroese exposure to these chemicals is also associated with the consumption of fulmar, a diet of sea mammals, fatty fish and perhaps eggs from seabirds and Puffin; is that correct?

A. That's what it says.

Q. Whale meat and blubber, seabirds and their eggs and Puffin are not typical components of the average diet in Vermont or New Hampshire, are they?

A. I think this is -- your statement is correct. This is a different diet.

MR. WOLFF: Mark this as Exhibit Number 8.

(Study was marked as Exhibit Number 8 for identification)

Q. Exhibit Number 8 is a study entitled Cohort Studies of Faroese Children Concerning Potential Adverse Health effects after mother's exposure to marine contaminants during pregnancy; is that correct?

A. Correct.

Q. And the only two co-authors on this study are Doctor Weihe and you; is that correct?

A. That's correct.

Q. On Page 4 in the first bolded heading, you wrote the -- strike that.

On Page 4, the first bold heading you quote the contaminants of the blubber adversely affect the immune system so that the children react more poorly to immunizations; is that correct?

A. That's correct.

MR. WOLFF: Mark this as Exhibit Number 9.

(Paper was marked as Exhibit Number 9 for identification)

Q. Exhibit Number 9 is a paper on which you are a co-author. First author is Kileman from 2010. Serum concentrations of antibodies against vaccine toxoids in children; is that correct?

A. That's correct.

Q. On Page 1434, the bottom of the middle column, you wrote that the Faroe Islands provide a unique opportunity to study PCB immunotoxicity given the PCP contamination of pilot whale blubber, a traditional food item favored by many Faroese, correct?

A. But not all.

Q. That's what you wrote?

A. Yes.

MR. WHITLOCK: But not all.

A. But not all.

Q. You concluded that developmental PCB exposure is associated with immunotoxic effect on serum concentrations of specific antibodies against diptheria and tetanus vaccinations. The immune system development during the first years of life appears to be particularly vulnerable to this exposure. That's what

you wrote; is that correct?

A. That's correct.

Q. And that is essentially the same hypothesis that you are advancing in this case except that here it concerns PFOA; is that correct?

A. I need to explain this, because when we wrote this paper, we had not yet measured the PFAS concentrations. So this is based alone on PCBs and we have not taken into regard the potential confounding from PFOA.

Q. On Page 29 of your report, Paragraph C, you describe reported findings from a Faroese birth cohort for concentrations of antibodies against antibodies and diptheria.

A. Point me to it?

Q. Paragraph C.

A. Okay. Can you repeat for me the question.

Q. Well, you described a study that reported findings from a Faroese birth cohort for concentrations of antibodies against tetanus and diptheria. Do you see that?

A. Yes.

Q. You don't provide a reference or end note for that study, do you?

A. It may not have, well, it is elsewhere in the report. It may not have been published at the time but it has been recently published. Before August 1, it was published.

MR. WOLFF: Mark this as Exhibit Number 10.

(Study was marked as Exhibit Number 10 for identification)

Q. Exhibit Number 10, is this the study that you are referring to?

A. Yes. That is the one.

Q. Good. In your report at Page 29, you write that the association support the notion that the developing adaptive immune system is particularly vulnerable to immunotoxic exposures to for example PFOA during infancy?

A. Yes.

Q. If you substitute PCBs for PFOAs as the example, and the notion that developmental exposure to PCBs can reduce vaccine responses in children, is essentially the same hypothesis that you are advancing with respect to PFOA here?

A. That's fine. There is no problem in having one compound having the same or overlapping outcomes as another one. So I don't see a problem.

Q. The Faroese marine diet is the source of the exposure to these chemicals; is that correct?

MR. WHITLOCK: Objection to the form. Vague.

A. With PFOA, if I may focus on PFOA the association with marine diet is not so strong. It probably comes from other consumer products and for example food wrapping materials because it does not occur in drinking water.

Q. So if there is no PFOA in the drinking water in the Faroe Islands, and the marine based diet is not the source of the PFOA exposure, how are the Faroese being exposed to PFOA?

A. Right. They expose exactly the same way as American, and Danes, and other Europeans in that PFOA is used for multiple purposes in society and the concentrations that the Faroese have are similar to what Americans have.

Q. In your 2010 study, you reported that decreased serum concentration of antibodies were attributable to PCB exposure, correct?

A. I need to make sure that you are talking about Exhibit Number 9.

Q. I am.

A. Okay. So I stand by that. I also said we did not measure PFAS at this time. When we found out that PFAS had the same affect and was much stronger, then we adjusted the PFAS association, that is, PFOA and the related compounds for PCB exposure. That is what is in my 2012 article in JAMA.

Q. In your 2010 study, Exhibit Number 9, on Page 1438 in the middle column, you wrote, the Faroese population is also exposed to contaminants other than PCBs such as PPDDE, hexachlorobenzene and beta hexachlorocyclohexane, because they correlate with PCB, this study could not assess whether they played any independent role; is that what you wrote?

A. That's correct.

Q. Exposure to PCB, mercury, and other chemicals is a potential covariate and confounder of any reported association between PFOA and a health-related endpoint in your study on the Faroese; is that correct?

A. Where do you have that wording from?

Q. I am asking a question.

A. This is your wording?

Q. Yes.

A. Let me then say that PCB would be a confounder based on the degree of association with PFOA, because if

there is no association, you will not have any conformity.

Q. Did all of your studies on PFAS exposure on the Faroese control for exposure to multiple PCB congeners?

A. I think we narrowed this down to total PCB. We did the calculations, also, for individual major congeners and the findings were the system.

Q. So it was not across the board?

A. You can't, because some of them are not detectable. So you just have zero exposure. And these statistics are not very reliable. If there is something that you cannot measure.

Q. Did all of your studies of PFAS exposure of the Faroese control for exposure to mercury, DDT, DTE, and hexochlorobenzene?

A. Not necessarily. Yes to mercury. We looked at it. There is no reason to test for confounding if it is not associated with PFOA exposure, and in this case, mercury was not associated with the antibodies. With DDE, it is a little more difficult because it is so closely associated with PCB, and if PCB is not a confounder, then the conclusion is that then PDE is not confounder.

Q. Did any of your studies of PFAS exposure on the

Faroese control for trans-nonachlor or beta-hexachlorocyclohexane?

A. It is not necessary to do so because we took into account PCB as the major pollutant because the exposures to those other substances that you are mentioning, particularly trans-nonachlor is much, much lower and highly questionable relevance.

MR. WOLFF: Off the record.

THE VIDEOGRAPHER: The time is 12:36. Off the record.

(Whereupon a lunch break was taken)

AFTERNOON SESSION

WITNESS RESUMED

THE VIDEOGRAPHER: Back on the record. The time is 1:29 p.m.

Q. Mr. Whitlock, counsel for the plaintiffs previously stipulated that the plaintiffs are not proffering you as an expert in medical monitoring. Do you recall that?

A. Right.

Q. Do you agree with that assessment?

A. Yes. Because it is in accordance with my introduction on Page 1.

Q. Is that because you are an expert in epidemiology and the substance and analysis in your report is addressed to epidemiology as opposed to medical monitoring?

A. No. It was just my understanding that what I have done here is what was needed and I was asked to challenge rebuttals from the three defense experts.

Q. So rather than addressing a medical monitoring program, your report speaks to epidemiological issues; is that correct?

A. And the need for medical monitoring. It certainly does but I did not delve into the details of

that.

Q. Let's return to the Faroe Islands.

A. Okay.

Q. Can you point to epidemiological evidence that PCB exposure is associated with an increase in the number of cases of diptheria in children in the Faroe Islands?

A. I don't think diptheria has occurred in recent years in the Faroes.

Q. Can you point to epidemiological evidence that PCB exposure is associated with an increase in the number of cases of tetanus in children in the Faroe Islands?

A. Again, I am not aware of any cases of tetanus in recent years in the Faroe Islands.

Q. Can you point to epidemiological evidence that PCB exposure is associated with an increase in mortality from colds or fevers in children in the Faroe Islands?

A. There may be such increase but I am not aware it is associated with PCB exposure.

Q. Now, I take it that since you not aware of any increase in the number of cases of diptheria, you cannot point to epidemiological evidence that PFOA exposure is associated with an increase in the number of cases of

diptheria in children; is that correct?

A. That's correct. If we are just looking at diptheria.

Q. Similarly, with respect to tetanus, you cannot point that epidemiological evidence that PFOA exposure was associated with an increase of number of cases of tetanus in children, is that true?

A. True.

Q. Can you point to epidemiological evidence that PFOA exposure is associated with an increase in mortality from colds or fevers in children in the Faroe Islands?

A. There is no such evidence as it has not been looked for.

Q. Do you agree that antibody responses of normal children to routine prophylactic vaccinations can vary substantially?

A. They can vary. That's correct.

Q. The reasons for this wide variation are poorly understood; true?

A. That is correct. It is not greatly understood.

Q. Do you agree that smoking during pregnancy can have long term adverse health affects on a fetus?

A. Do you mean beyond birth or do you mean during

gestation?

Q. Beyond birth.

A. Beyond birth. I agree, so, yes.

Q. Would you agree that a person's genetic make up plays a substantial role with regard to health and longevity and mortality?

MR. WHITLOCK: Objection to the form. Vague.

A. This is correct. Genetic make up may even make a subject more vulnerable to PFOA but we don't know the detailed evidence yet.

Q. The Faroe Islands are by definition insular; is that correct?

A. Yes. The Faros are a number of islands in the North Atlantic.

Q. And the Faroese are a fairly homogenous population; true?

A. Yes. This is what I have claimed at least.

Q. In your opinion, these factors make the Faroese an advantageous study population; is that correct?

A. This is what I have written, yes.

Q. Is it fair to say that you believe that this makes the reported findings on developmental exposures to be generalizable to other populations beyond the

Faroe Islands?

A. I believe so. Yes.

Q. The Faroese are a fairly genetically homogenous population; is that true?

A. That's what I understand.

Q. And an isolated and genetically homogeneous population can be particularly susceptible to higher rates of heritable disorders; is that correct?

A. Yes. To some degree.

Q. And the frequency of these heritable disorders in the Faroe Islands is unlikely to be similar to that in Vermont or New Hampshire; is that correct?

A. Right.

Q. And the Faroese are predisposed to primary carnitine deficiency?

A. Yes. That has been discovered.

Q. Carnitine is found in the acidity of fatty acids and glucemic regulation; true?

A. I am not sure how important it is in regards to glucose metabolism, but certainly with regard to fatty acid.

Q. Do you agree when the body is unable to oxidize fatty acids, that fats accumulate in the liver, skeletal muscle and the heart?

A. I would have to look it up. I believe this is correct.

Q. Liver steatosis also has an affect on the liver's failure to properly metabolize fats; is that true?

A. I think so.

Q. Did your studies of the Faroese control for the incidents of carnitine deficiencies?

A. Yes. To the extent those cases were identified we did not include them in the study.

Q. Are you aware that researchers have reported that the Faroese have the highest prevalence of glycogen storage disease type 3A in the world?

A. I don't remember. It is likely true.

Q. Isn't it true that beginning in infancy, individuals with any type of glycogen storage disease type 3 may have low blood sugar, excess amounts of fats in the blood, and elevated blood levels of liver enzymes and that as they get older, children with this condition typically develop an enlarged liver?

MR. WHITLOCK: Objection to the form.

A. I think what you are saying is correct. It is not relevant to my studies.

Q. Are you aware of any statement in the peer review scientific and medical literature which reports PFOA is

a cause of kidney cancer?

A. Not in the Faroes but in general?

Q. In general.

A. It is a cause, I think the wording that has been used is risk factor.

Q. And a risk factor is not synonymous with a cause, is it?

A. It depends on the circumstances. Because in scientific literature, we don't necessarily use the word causation but use a risk factor as something that we need to adjust for when we consider potential confounding.

Q. Is cigarette smoking a risk factor for cirrhosis of the liver?

A. I don't remember. It may well be.

Q. Does cigarette smoking cause cirrhosis of the liver?

A. Again, I can't give you an answer because it is not something that I remember.

Q. Isn't it true that people who tend to drink large amounts of alcohol also tend to smoke cigarettes and for that reason cigarette smoking is a risk factor for cirrhosis?

A. I would not use that wording. I would say that

it is a potential confounder.

Q. Are you aware of any statement in the peer review, scientific and medical literature which reports that purports PFOA is a cause of testicular cancer?

A. Again, it may well be written somewhere in the literature. I do not remember it. I would think the wording used is a risk factor.

Q. Are you aware of any statement in the peer review scientific and medical literature which reports that PFOA is a cause of steatohepatitis?

A. Again, I don't remember.

Q. Are you aware of any statement in the peer review scientific and medical literature which reports that PFOA is a cause of any particular disease in humans?

A. Again, I can't answer you. I don't remember.

Q. You would agree that every individual has his or her own background risk of developing the diseases that you describe on Pages 27 through 62 of your report; is that correct?

A. Right. We all have different background depending on age, and sex, other factors.

Q. Hereditary factors can contribute to an individual's background risk; is that true?

A. I agree.

Q. And an individual's level of physical activity can affect their background risk for some health endpoints; is that correct?

A. That's correct.

Q. And eating habits affect and individual's background risk as well, true?

A. It depends on the outcome. I would agree in general for some outcomes, yes.

Q. The individual's age at a given point in time is also a factor when evaluating one's background risk; is that true?

A. I would agree.

Q. Please assume with me that there are two individuals. Individual Number 1 is a forty-year old male. Never a smoker. Individual Number 2 is a forty-year old male, a pack a day cigarette smoker since his teen years. What would you estimate is the approximate relative risk of non small cell lung cancer of Individual Number 1 versus Individual Number 2?

A. I can't give you that number. I would have to look it up.

Q. Why can't you give that number?

A. Well, because I don't remember it. You can have a small cell lung cancer if you are a non-smoker. Those

cases occur. It could perhaps be attributed to indirect smoking. It could be perhaps effected by occupational exposure, could be effected by genetic susceptibility. So that risk, who's non-smoker is not necessarily zero or close to zero, but certainly everything else being equal, the smoker would have a higher risk.

Q. How would you know that?

A. How would I know that? I would rely on the scientific literature and my judgment.

Q. Are you referring to epidemiological studies or something else?

A. No. This is based on epidemiology and I would say the assessment of IARC.

Q. Please assume that a 65 year old man is diagnosed with non small cell lung cancer and that he has a forty year pack cigarette smoking history. Besides tobacco smoking, what are the other possible causes of his lung cancer?

A. Again, I don't remember the details. So I would have to look it up.

Q. Can you give me any possible other causes?

A. Not at this point. I have not looked at that literature lately.

Q. Would an unknown cause be a possible cause of his

lung cancer?

A. Again, I have to make some assumptions. So I can't really give you a good answer today.

Q. What would be the most probable cause of this individual's lung cancer?

A. The forty pack a year -- pack a day smoker.

Q. So the sixty-five year old man, forty pack year smoking history?

A. Forty pack years?

Q. Yes. Not forty packs a day.

A. Okay. You are asking me to make assumptions. I would rather not answer the question.

Q. Well, you understand that as an expert witness, I can ask you to make assumptions?

A. That is fair enough. Let's stay with the topic that is related to my expert report.

Q. Please assume that a 65 year old man who has been a metal worker for forty years develops COPD. Please further assume that OSHA and state agencies have repeatedly cited his employer over the term of his employment for violating occupational exposure standards for mineral fumes, welding fumes, and sulfur dioxide, what would you say are the possible causes of his COPD?

A. Again, I have to reserve my judgment. You are

giving me personal information and you are saying that an agency has cited the employer, but I don't know what the basis of that citation and I don't know what the levels of exposure are and what, how they are compared to regulatory guidelines. So I can't answer that question.

Q. Other than the occupational exposure to the substances mentioned, what are the other possible causes of his COPD?

MR. WHITLOCK: Objection. Calls for speculation.

A. Yes. Certainly smoking is a risk. I am sure that there is a genetic component as well. I would like to limit it to that because I have not prepared for questions like this.

Q. Is an unknown cause also a possible cause of the COPD?

A. Unknown causes are always possible until they have been discovered because then they turn to be known risk factors.

Q. What is the probability that his COPD is due to a cause other than occupational exposure?

A. I would refer to my answer before that I have not prepared to answer questions like this.

Q. Please assume that there are two individuals. Individual Number 1 is a 25 year old female with a BMI of 31 living for the past three years in a town where the PFOA level in the drinking level is 70 parts per trillion. Individual Number 2, 25 female with a BMI of 31 with no known exposure to PFOA. What would you estimate is the approximate relative risk of developing Type 2 diabetes in Number 1 versus Number 2?

MR. WHITLOCK: Objection to the form.

A. Again, I am looking at this from a population viewpoint. I am not looking at individual risk. So I can give you information about epidemiology as we have looked at female nurses in America and I'm sure some fom Vermont in regard to their BMI and their blood concentration of PFOA; but I would have to look up that information because I don't remember the details on 25 years. And BMI of 31. I can't just offhand give you a number.

Q. Please assume that one year later, individual Number 2, that is the woman with no known exposure to PFOA develops Type 2 diabetes. What are the possible causes of her Type 2 diabetes?

MR. WHITLOCK: Objection to the form.

A. Let me just say that genetic predisposition is a

known factor. And when we are talking about individuals, we can't easily refer to particular causation, but of course in this case if there is a family history for Type 2 diabetes, it is obvious that must somehow have contributed.

Q. Are there any other possible causes of her Type 2 diabetes?

A. Not that I wish to discuss right now.

Q. Is an unknown cause a possible cause of her Type 2 diabetes?

A. I refer to my previous response that unknown causes is always possible until you find out what they are and then they turn out to be known causes.

Q. What is the most probable cause of her Type 2 diabetes?

MR. WHITLOCK: Objection to the form.

A. This all depends on sex and age. I am not prepared to give you a complete discourse on that.

Q. So here we have a female who is 26 years old.

A. What about it?

Q. Well, I have just given you the sex and the age.

A. Right. What's your question? I don't understand.

Q. What's the most probable cause of her Type 2

diabetes? A 26 year old female with a BMI of 31 with no known exposure to PFOA.

MR. WHITLOCK: Objection to the form.

A. Given that the BMI is elevated, I think it would probably had a role in the causation. That's all I can say at this point.

Q. Please, assume that five individuals, let's call them A, B, C, D, and E, are males, all of the same age, and BMI, who have lived in a town with the PFOA level in the drinking water is seventy parts per trillion. In your opinion, do all five have the same probability of developing ulcerative colitis or thyroid disease or kidney cancer or hypercholesterolemia or testicular cancer?

A. From the information given to me, I would say yes.

Q. Why is that?

A. Because they have exactly the same characteristics.

Q. Please assume that subject -- strike that.

How do you know they have the same characteristics?

MR. WHITLOCK: That's what your question said.

A. Yes.

MR. WHITLOCK: You are asking him to make assumptions.

MR. WOLFF: Jamie, if I wanted to hear your answer, I would swear you in.

A. But that was my answer.

Q. My question to you is, how do you know that five different individuals are otherwise identical in all respects?

MR. WHITLOCK: Because you didn't ask him to make that assumption.

MR. WOLFF: Jamie, I am not asking you the question.

A. Well, let me answer it. You have only given me this limited information and it is all the same. So I have to assume that you want my answer based on that and my answer is the same risk.

Q. In your experience, are all individuals identical from individual to individual?

A. Okay. We have to make something clear here. You are talking about individuals, either one, two, five. I am an epidemiologist. I look risk which is population based. I am responding to you on the basis of human populations. Not on individuals.

Q. So your analysis here would be an epidemiological analysis because you deal with populations of people as opposed to a clinical medicine prospective which deals with individuals, is that fair?

A. That is fair.

Q. Please assume that Subject A develops ulcerative colitis. What are the causative or predisposing factors known for ulcerative colitis?

A. Very little is actually known.

Q. Of those factors that are known, what are they?

A. I mean, age is a factor because it is a disease that often times develops in young people. But then there is as far as I remember a second peak later on in life where the incidents increases again. So it is age dependent. Whether there is a difference between men and women, I don't remember. I don't think it is very big. There may be one.

Q. Anything else that you would put on the list?

A. Well, I mean, one of the issues that is currently being researched is the microbiome. There have been done, studies have been done of changing the microbiome with fecal transportation. So it looks like this are aspects of microbiome that plays a role for the causation of that disease. But what they are is

currently unclear.

Q. Anything else that you would put on that list?

A. I believe that there is a genetic component as well. That's all I can remember at this point.

Q. Of those factors, which is the highest probable causative or predisposing factors?

A. Again, I would have to look that up.

Q. What is the probability of PFOA as the causative factor?

A. I believe that there is substantial evidence that it is.

Q. What is the probability numerically?

A. We don't know exactly what the probability is. It is like a judgment call that the evidence is substantial; but I do agree that we should try to get more research on this and I am doing research on this. So I hope that we can contribute some more evidence.

Q. Are any other chemical or physical agents potential causes of ulcerative colitis?

MR. WHITLOCK: Objection to the form.

A. Again, I would have to look it up.

Q. Sitting here today, you do not know, is that fair?

A. Yes. That's fair enough.

Q. Please assume that Subject B develops hypercholesterolemia, what are the predisposing factors known for hypercholesterolemia?

A. First of all, there is a genetic, a mutant that predisposes to what is called familial hypercholesterolemia that is easily recognized because the concentrations are really, really high. So that's a genetic component. There are obviously dietary and physical activity aspects of that.

Q. Sure. Of those factors, what is the highest probable causative or predisposing factor for the hypercholesterolemia in Subject B?

MR. WHITLOCK: Objection to the form.

A. The point is that genetic can be very strong and it is not that it is very common, but it is just that when you see it, the concentrations are so high. So that's a strong factor.

Q. What is the probability of PFOA as the causative factor?

A. Again, I think the evidence is substantial.

Q. Can you quantitate that?

MR. WHITLOCK: Objection to the form.

A. What one can do, but it has not been done yet, what, a meta analysis of the data that are available,

but not all of the study are as well conducted as they should. So one would have to go into detail to assess the, you know, variety of studies that are available. There are several.

Q. Do you know how to conduct a meta analysis?

A. I know and I have done that in the past, but I didn't think is that I should spend my time doing that for this purpose.

Q. So just to be clear, you have not performed a meta analysis with respect to PFOA exposure and any health endpoint; is that correct?

A. That's correct.

Q. Are there any other chemical or physical agents that are potential causes of hypercholesterolemia?

A. I believe so. I don't remember any of them.

Q. Please assume Subject C develops thyroid disease. What are the causative are predisposing factors known for thyroid disease?

A. I mean, there are several types of thyroid disease. Certainly, there are factors such as radiation. Say it is radioactive iodine, that's certainly a risk factor that we are very much aware of. Particularly in the Chernobyl area.

Q. What are the other predisposing or causative

factors besides radioactive iodine?

A. I mean, I teach this. I usually use a biomechanical depiction of what happens in the thyroid gland and then I have a variety of chemical compounds. How they interfere with this iodine, it interferes with the enzyme and that. Dioxin is one. There is some pesticides that interfere.

Q. What pesticides?

A. I would have to go back and refer to that. I did not prepare for that today.

Q. Of those factors, which is the highest probable causative or predisposing factor?

A. You know, that depends on the level of the exposure. Dioxin is a risk factor. Dioxin exposure has gone down. So I doubt that dioxin any longer is a major factor. Likewise with radioactive iodine far from Chernobel.

Q. What is the likelihood that the thyroid disease is idiopathic?

A. The word idiopathic means that there is no known cause of it. It is a difficult word that physicians use when they don't know what to say. So the word idiopathic means thyroid disease of unknown cause. It is not very helpful.

Q. What is the likelihood that the thyroid disease has an unknown cause?

A. I think that's very substantial.

Q. What is the probability of PFOA as the causative agent?

A. Again, I refer to thyroid disease in my report and I think I used the word substantial evidence.

Q. Can you give me a point estimate for the relative risk?

A. No, I can't. Because that type of detail does not yet exist.

Q. Please assume that Subject D develops kidney cancer. What are the causative or predisposing factors known for kidney cancer?

A. Well, I should say that the most common form of kidney cancer, and I think that's what you refer to hyperlymphoma. Then we would have to refer to that and note metastatic cancer. Anyway, that disease is to a substantial degree of unknown origin. There is very little occupational, for example, evidence of particular chemicals causing hyperlymphoma.

Q. What's the probability PFOA is the causative agent?

A. I have the same answer again. That there is

substantial evidence with the exact probability in part unknown and in part also depends on the subject who gets the cancer. Because other factors may play a role as one age. That is the only way I can answer that question.

Q. Finally, let's talk about Subject E. Assume that Subject E develops testicular cancer. What are the predisposing factors known for testicular cancer?

A. Well, there are different forms of testicular cancer. At least there is one form that occurs at a younger age and then picks up later on in life. Although we don't know the details yet, it looks like. So we do not know the details yet. There is at least one prominent factor that has been researched in recent years and that is inter uterine exposure to endocrine disrupters, particularly androgen disrupters

Q. Is that some sort of an endocrine disorder?

A. No. It is because when the fetus is like eight or nine weeks, that is when the sexual differentiation begins. Otherwise, all humans will become women. So there has to be the correct imprinting happening and for the male it is that testicular cells develops so they can generate testosterone that then tricks that development. If there is some interference, and we

don't know if PFOA does it, but it is possible if that interference happens from an exogenous cause, then s that can cause abnormalities which include testicular cancer.

Q. What is the probability of PFOA as the causative agent?

A. I believe it is substantial as I have written in my report.

Q. Can you give me a relative risk point estimate for that?

A. Again, I have to refer to my previous question that that depends on the circumstances, the age, level of the exposure; but there is no doubt, well, I will say there is substantial evidence that testicular cancer is an effect that can be attributed to PFOA.

Q. What is the relative risk range?

A. Again, I can't tell you what the exact relative risk is.

Q. I'm not asking exact. What's the range?

A. I can't. I would have to sit down and look at the exposure levels and do some calculations. I can't do that on the spot.

Q. Is there a methodological difference between identifying risk of an adverse effect from an

epidemiological study and identifying the cause of an adverse event in a clinical trial?

A. There is. In the clinical trial as I understand it, it is a controlled trial with blinding, where you control the exposure. So we are back to the question you asked before that we essentially in the clinical trial conducting an experiment where we know exactly the dosage, but that's not what we do in exposure studies. For example, of the Vermont population exposed to PFOA, where we can only evaluate the exposure after the fact.

Q. Are there any other methodological differences between identifying a risk of an adverse effect from an epidemiological study and identifying the cause of an adverse event in a clinical trial?

A. I think we already touched upon the major issues. If there is anything specific, I am happy to comment on that.

Q. I want to make sure we are not leaving anything on the table. Thanks.

The studies that you cite on Pages 27 through 62 of your report are not the totality of the published studies from PFOA exposed human populations on each of those endpoints, are they?

A. No. They are, of course not, all of the studies,

those are the major studies.

Q. What were your inclusion and exclusion criteria if any for the studies that you cite on Pages 27 to 62 of your report?

MR. WHITLOCK: Objection. Asked and answered.

A. What I did was to look through the complete evidence essentially what I had access to. There may have been some in languages that I don't speak that I have missed, but I looked at the accessible literature and I also looked at FSA and ATSDR, and what I did was to select the literature that I thought contributed to the evaluation of a possible association of the outcome with PFOA exposure.

Q. In your opinion, how long would a person have to drink water containing PFOA to be at an significantly increased risk of a serious latent disease?

MR. WHITLOCK: Objection to the form.

A. Right. So you are not giving me the full information, because I would ask for the PFOA concentration in the water. I think that's very important to judge whether there is more than a background risk. So what we talked about here, PFOA exposure from the drinking water in the class, I assume

this is what we talked about, well, because any way, and we talked about residents. So they are not just having exposure during their vacation, but they are residents. We are talking about longer term exposure and that totality in my mind adds to a clearly elevated risk, elevated exposure, I should say. Therefore an elevated risk.

Q. In your opinion, how long would a person have to drink water containing 70 parts per trillion of PFOA to be at an significantly increased greater risk of a serious latent disease?

A. I am not prepared to answer that. Because I would want to make some calculations before I answer.

Q. In your opinion, how long would a person have to drink water containing 140 parts per trillion of PFOA to be at increased risk?

A. The same answer.

Q. If I was to ask you five or ten or one thousand parts per trillion, your answer would still be the same; is that correct?

A. It would because it is all on a relative scale. I think this is a question that has to be settled by the court. It is not my medical opinion that is important here.

Q. Sorry. What's the issue that has to be decided by the court?

A. If a subject who has, if a subject has to have an exposure of a thousand like you mentioned in order to be approved as a member of a class by the court, that's not up to me.

Q. That's not what I am asking. I am not asking you for who is included in a class definition or not. I am asking from a medical and scientific perspective. Not a legal one.

A. Right. Okay. I can then give you a flat answer which is sort of very general. That is my opinion. That is does all of the water in that area contain a toxic and manmade substance that has nothing to do there. According to my calculations, I need to have very, very low water levels, below one, as we discussed before in order to be safe.

Q. I appreciate that you discussed that. My question is a little bit different.

How long would a person have to drink water containing PFOA, whether it is at five parts per trillion, ten parts per trillion, a hundred parts per trillion or a thousand parts per trillion to be at a significantly increased risk of a serious latent

disease?

MR. WHITLOCK: Objection to form. Calls for legal conclusion.

A. Again, I am a scientist. I have to refer to what I have studied and what I have read myself. I have done the calculations saying that the levels should be below one and now you mentioned to me some very high levels in comparison in asking me to say how long does it take. I can't do that calculation offhand.

Q. How long would a person have to drink water containing one part per trillion of PFOA? In your opinion, how long would a person have to drink water containing one part per trillion of PFOA to be as a significantly increased risk of a serious latent disease?

MR. WHITLOCK: Objection to form. Calls for legal conclusion.

A. Again, I have suggested that the level should be below one. Now, you are giving me a number just before that and I think that it is a theoretical issue of what the actual risk may be. It is clear that it will be less than at seventy but it is not zero.

Q. How long would a person have to drink water containing just less than one part per trillion of PFOA

to be at a significantly increased risk of a serious latent disease?

MR. WHITLOCK: Objection.

A. I think I answered this in various ways. I have to refer to my previous answer.

Q. If there is a risk associated with drinking water with PFOA, then that risk is different from individual to individual because their consumption of water is different; is that correct?

A. The consumption level, well, of course, in fact the uptake at the same concentration in the water and then that would then also relate to differences in risk.

Q. Assuming there is a risk associated with PFOA exposure, if you were to look at any individual putative class member among the thousand or more individuals in this proposed class, the risk to that individual is going to be affected not only by the amount of water but also by the amount of PFOA in the water he or she has consumed; is that correct?

A. Exactly.

Q. Is it fair to say that the risk of disease if any is not proportional to the difference in the amounts of water consumed because different individuals have different susceptibilities to PFOA based on their

medical conditions or other behavioral factors?

A. I would not express it that way. I would say that of course there are factors such as sex and age that play a role, but I am looking at this from a population viewpoint, and the greater the exposure, the greater the uptake, the greater the blood concentration, the greater the risk. That is my best answer to that question.

Q. Is it fair to say that individuals who are not exposed to PFOA through their home's water supply because for example they are hooked up to the municipal water system in Bennington or because they have activated carbon filter systems that they passed their water through, do not have that component of PFOA in their body burden and thus their risk of exposure if any is less than those individuals who have PFOA in their home water drinking supply?

A. And they are drinking the same amount about, and, yes. Of course, I mean, again, I have to give you my same answer. The risk is not nil but clearly there are relative differences.

Q. Is an alteration of a biochemical marker itself a clinical pathology?

A. This is a matter of judgment. This is something

that regulatory agencies are very much into. As a scientist, it is a whole lot easier to look at markers because everybody has an enzyme at some level or whatever that mark is, but it is not everybody that has fatty liver disease.

Q. In your opinion, are small but statistically significant increase in liver biochemical tests clinically significant?

MR. WHITLOCK: Objection to the form.

A. It depends. If it occurs, you can discuss that without the reference interval or if it is high, let's say, a young person or a child, it may be more significant than if it perhaps is for an elderly person who has already been diagnosed hypercholesterolemia. It is really, I have to mention caveats like that.

Q. In your opinion, does every instance of an abnormal liver biomechanical test constitute a disease?

A. I can't think of an example that it does, because in my, according to my knowledge of clinical matters in a diagnosis is never, oh, I can't say that because that is not true. A clinical diagnosis would rely on some type of clinical findings that are usually beyond just clinical pathology block tests.

Q. So would you agree even if an individual's liver

panel test results are different from the normal value, that the individual may not have a problem?

A. Again, that's hard to say, because it could perhaps be some genetic or constitution that makes this person to be high in particular liver enzymes. I mean, I would not as a physician rely on that solely.

Q. What else would you rely on?

A. I would rely for example on a liver biopsy, on a scanning. I would rely on a whole battery of clinical tests. You can also, you can assess crudely the size of the liver.

Q. Is there any risk of performing a liver biopsy?

A. There is of course a risk associated with that. So this is what you like to do when there is a substantial likelihood that there is something to find. This is not something that I would ever do in a population study of a healthy subject.

Q. On Page 28 of your report in the fifth paragraph. You write, my review of available epidemiological studies demonstrates a strong link between PFAS exposure and adverse effects on human immune system functions. Do you see that?

A. Yes.

Q. What is a strong link?

A. Well, it is another way of saying that there is substantial evidence.

Q. Is the phrase strong link synonymous with a cause and effect relationship?

A. It is pretty close. It certainly is stronger than a possible link that we talked about before.

Q. It is close but not quite?

A. This is a matter that would have to be discussed with, you know, whoever the recipient is. If this is the US EPA, for example, if they would regard the evidence strong enough to call it causal, and I would not preclude what EPA would decide on that.

Q. As you use the phrase strong link, are you using it to denote a causal relationship?

A. I didn't use that word. I am saying that there is substantial evidence. That is as close as I can get at this point.

Q. Doctor Ducatman is not proposing monitoring of serum antibodies or any immuno globulin concentrations, is he?

A. I think you are correct. I don't remember it by heart.

Q. Doctor Ducatman is not proposing monitoring for immune toxicity or immuno suppression, is he?

A. Again, my answer is the same. I think you are correct.

Q. On Page 34 in Paragraph E of your report, you write I conclude that the human evidence strongly supports the existence of PFAS dependent immuno toxicity background exposure levels. Correct?

A. That's what I said here.

Q. Is it your opinion that the average American is experiencing immuno toxicity as you term it?

A. I would say so.

Q. Is an alteration of an immunological biomarker itself a clinical pathology?

A. It depends on what you consider clinical pathology. I consider it a marker of immune dysfunction where we don't quite yet understand the reach of that dysfunction to which degree this, for example, might be linked to greater cancer risk. Because the immune system is involved in removing abnormal cells. We don't know that yet.

Q. We don't know?

A. This is what IARC says, is this the possible mechanism. We don't know the mechanism for PFOA to what causes cancer. But it is possible that the immune system could play a role.

Q. Is it fair to say for example that a reduction in an individual's diptheria antibody concentration does not mean that individual will necessarily contract the diptheria?

A. That's correct. But you have also to consider that we did not look at that antibody as such. We looked at the T cell dependent B cell function and plasma cell generation of the specific antibodies. So as an integrated function of the immune system, what we found in the immune system was deficient. So we were not interested in diptheria.

Q. Let me ask you a couple of questions. I think we may have covered this before. If we have, I apologize.

A. No problem.

Q. In the last paragraph on Page 10 of your report, when you make reference to Bennington residents who have an accumulation of PFOA in the body at above background levels, you are referring there to residents who exceed 2.1 micrograms per liter, correct?

A. That's what I say here.

Q. How can one tell which Bennington residents do or do not have more than 2.1 micrograms per liter of PFOA in their blood?

A. I can only guess, but I took this information

from the case definition or rather, what is it called, the class definition.

Q. So my question is a little bit different than that.

Looking at the Bennington population at issue, which are some one thousand or so individuals, how can somebody tell whether any particular residents do or do not have more than 2.1 micrograms per liter of PFOA in their blood, how would one know that?

A. You are asking an epidemiologist, and I would say it is a great majority of those people who would have more than 2.1.

Q. How would one determine that?

A. I would assume that, I mean, this is not, I have not been into this. The way to figure that out is to take a blood sample and analyze it.

Q. Fair enough. We touched upon this very briefly before. You are aware that NHANES periodically conducts and publishes results of blood sampling; is that correct?

A. I understand that very well.

Q. Is it fair to say NHANES collects and publishes data on human exposure to some 265 environmental chemicals?

A. I don't remember the exact number, but that sounds right.

MR. WOLFF: Mark this as Exhibit Number 11.

(PFOA blood data tables was marked as Exhibit Number 11 for identification)

Q. Exhibit 11 contains the PFOA blood data tables from the February 2015 NHANES fourth national report on human exposure to environmental chemicals. If you would, please, turn with me to Page 338 which contains the sampling results for PFOA from 2011 to 2012. Are you there?

A. Yes.

Q. Top line, the geometric mean for the total sample is 2.08, which rounded up to the first decimal is the number that you have been using and we have been using, which is 2.1 micrograms per liter; is that correct?

A. That's correct.

Q. If you would please turn to Page 336, which contains the sampling results for PFOA from five different surveys taken between 1999 and 2010; is that correct?

A. Yes.

Q. With the exception of the 2007 to 2008 sampling, in each successive survey, the geometric mean is going

down from a prior survey; is that correct?

A. That's correct.

Q. For 1999 to 2000, the geometric mean was 5.21 micrograms per liter; is that correct?

A. Yes.

Q. As we know, for 2011, 2012, the geometric mean was 2.08; is that correct?

A. Yes.

Q. Since you say that anybody in this class who has PFOA blood serum levels above 2.1 would require medical monitoring, would you say that millions and millions of Americans should have been getting this medical monitoring as a medical necessity in 1999 to 2000 when background level was 5.21 which is two-and-a-half times higher than 2.1?

MR. WHITLOCK: Objection to the form.

A. I have to correct you because I am not saying that. You should refer to that paragraph that you referred to before on Page 10. The bottom paragraph. Because I am referring to the class information. That's not mine. What I am saying is that this population is and has been exposed to an elevated exposure and therefore elevated risk and because it is under those local circumstances, I am saying that elevated risk

should trigger medical monitoring. So I am not talking about all the United States here.

Q. Let me ask you this. Would you say that millions and millions of Americans should have been getting this medical monitoring as a medical necessity in 1999 to 2000 when the background level was 5.21 micrograms per liter?

A. I have never considered that question, but I can tell you that one of the reasons is that these amounts, these concentrations and serum is so high is that we know water has been contaminated across the United States on numerous sites and we know that at least in one of the studies that we did, five or six million Americans are exposed to elevated concentration of PFASes in drinking water. So parallel to the Vermont case and those numbers are included here. That's why this is not really a proper background. It is actually too high. Maybe it was even much too high in the past.

Q. I guess my question is, if anybody in a population has got 5.2 micrograms per liter of PFOA in their blood, would you say that they need medical monitoring as a medical necessity?

A. I would not say that because that depends on the circumstances. I understand the circumstances here are

quite specific in that there is a local source, and we know exactly comes through drinking water. We know these are resident who have been exposed to this for quite a while.

Q. If the exposure level is the same from an individual in Bennington and an individual in Peoria, Illinois, they have the same identical blood levels, what difference does that make as to whether they require medical monitoring for potential future disease?

A. I can't answer that. Medical monitoring in this particular case has been defined as it is here and all I am saying that is reasonable. I can support that. And the way that Doctor Ducatman has described it, I am in favor of that. I have not been asked to look the state of Illinois and specific locations. I would give you that those elevations and serum concentrations will result necessarily in an increased risk. That relates to my expert report here.

Q. I guess what I am sort of having trouble getting my head around is if a person has got 5.1 micrograms per liter of PFOA in his or her blood in California or Arizona or Texas or Missouri or Mississippi or New Jersey or Vermont or New Hampshire, why should the particular location where that person resides make a

difference from a clinical perspective as to whether they need medical monitoring for exposure to PFOA?

A. I completely understand your question. All I can say, I didn't comment on medical monitoring. I did look at Doctor Ducatman's report and I think he argued that convincingly. I have not seen similar considerations on other locations in the United States. I do insist that they have an increased risk as well, but whether that should trigger perhaps a completely different circumstances of medical monitoring, I have no opinion.

Q. Have you ever expressed an opinion that any particular population requires a medical monitoring program?

A. I don't remember so in this regard, I don't think so.

MR. WOLFF: Mark is this last Exhibit Number 12.

(Transcription was marked as Exhibit Number 12 for identification)

Q. So Exhibit 12 is a transcription of the ATSDR, CDCs January 2017 PFAS continuing education for clinicians, which is available on video through the ATSDR's website, and which contains subtitles. I just want you to be clear about this document. This document

was prepared by our word processing department using the transcription off of that video and its subtitles.

A. Okay.

Q. Please, turn with me to Page 28. As recently as 2017, the ATSDR, CDC has told clinicians that there is no health screening recommended because of exposure to PFOA; is that correct?

A. I see that.

Q. Please, turn with me to Page 27. We are going to take this in stepwise fashion. As recently as 2017, the ATSDR and CDC has told clinicians that PFAS are ubiquitous in both the United States and globally. There are no specific biomarkers of health affects caused by or linked to PFAS blood concentrations. The presence of PFAS in blood testing only confirms exposure which is present and greater than 95 percent of the United States population based on representative samples from the NHANES studies; is that correct?

A. I see that.

Q. Do you agree with that statement?

A. I agree that PFAS is ubiquitous in the United States. It says there are no specific biomarkers of health effects. I wonder what they referred to. It just says biomarkers. I would like to know what

biomarkers they refer to. And the presence of PFAS in blood confirms exposure. Well, that is a standard expression from government or authorities. While higher blood --

Q. I am getting there next. I did not get there yet.

MR. WHITLOCK: What page are you on?

MR. WOLFF: 27.

Q. Let's take that next statement, Doctor. The ATSDC, CDC further state, while higher blood concentrations of PFAS express larger exposure, PFAS blood concentrations cannot be linked to any specific health effects and results obtained from testing patients, blood PFAS concentrations would not guide medical decisionmaking; is that correct?

A. I see that, but you have to recognize that that is a statement on behalf of the Centers for Disease Control, which is always trying to, you know, provide a statement that cannot be upsetting to anybody. This is a type of statement that they will say about fluoride in drinking water. This is just their interpretation which has, you know, different, defensive, so to speak aspects in addition to the science. It is not just a pure summary of the science.

Q. Let's go to the next statement. The ATSDR CDC further states that even if a patient is identified as having an extremely high PFAS blood concentration, this does not mean he or she will suffer from any adverse health affects; is that correct?

A. That's what it says. It is sort of a strange statement.

Q. Do you agree with that statement?

A. I can agree in the sense that we talked about before that on average, the risk is higher. But the fact that there is an elevated risk does not mean that a specific individual will develop specifically that disease.

Q. The ATSDR, CDC further states that, likewise, patients with mildly elevated PFAS blood concentrations are not immune from exposure related health risks. Management of patients exposed to PFAS should be guided solely by patient symptoms and findings derived from a thorough health history and physical examination; is that correct?

A. It is correct that that's what it says.

Q. My next question is, do you agree with that statement?

A. I agree with it to the extent that I would take

into account the PFAS exposure.

Q. Do you agree with or disagree with anything else in that statement?

A. This very last, the management?

Q. The very last one.

A. I have difficulty with the one where they say the management should be guided solely by the patient's symptoms and findings deriving from a thorough physical health history and physical examination. That is not enough. For example, that could be physician reports on something else and that's not just a health history. That could be occupational exposure. Similar things. I would not, what you said, this has been taken off a tape. I think this is not a proper complete statement and I don't think CDC would give you that impression that it is.

Q. Please turn with me to Page 32. Are you there?

A. I am there.

Q. The atsdr CDC states that more than 95 percent of the represented United States population has measurable blood levels of PFOA and PFOS. The presence of these PFAS in blood only confirms exposure. This does not mean your patient will suffer any adverse health effects. Routine blood test for PFAS cannot be

extrapolated to any specific health effects and cannot guide medical decisionmaking; is that correct?

A. That's what it says.

Q. Do you agree with that statement?

A. No. In the present situation, Number 1, we know more than they knew when they wrote this up, and what they decided to say on that occasion later on in January two years ago. So that's one aspect. I think that, I mean, the statement that your patient will not necessarily, they are missing the word necessarily, suffer from any adverse affects. That has to be interpreted properly in that people who have elevated exposures to PFAS have necessarily an elevated risk, and in the situation in Vermont where you have the zone of contamination, you have a confined population. This is where we have been arguing about medical monitoring.

Q. Is it fair to say that because of biomonitoring sampling results for PFOA cannot predict current or future health outcomes or diseases, the results are not clinically actionable?

A. Again, I would say, we should ask CDC if that is our view today, because it does not sound right that CDC would issue that as guidance to physicians of this country. They should know better today. Also, given

the most recent ATSDR report.

MR. WOLFF: Let's take a break. Off the record.

THE VIDEOGRAPHER: The time is 2:45 p.m. We are going off the record.

(Whereupon a break was taken)

THE VIDEOGRAPHER: We are back on the record. The time is 2:56.

Q. So during the break, counsel had a discussion sort of flush out the parameters of the stipulation from the plaintiffs' counsel. So while we understand that it has been stipulated that the plaintiffs are not proffering you as an expert in medical monitoring, and as a result of that, we are going to forego questioning about individual blood tests, specificity, sensitivity, positive predictive value in the population, that you are nevertheless endorsing as a general principle Doctor Ducatman's recommendation for a medical monitoring program, at least conceptually; is that correct?

A. It is reasonable and pragmatic and I think justified.

Q. Is it fair to say that medical monitoring in the form of medical screening is a systematic search for a disease in people who have no signs or symptoms of that

disease?

A. I think that is reasonable.

Q. When you commit asymptomatic patients to medical monitoring, you are making a medical decision and are intervening in their lives medically; true?

A. It depends on what you think to intervene. It is a medical intervention. But the degree to which it intervenes with peoples' lives is a matter of judgment.

Q. In an asymptomatic population, is it fair to say that the vast majority of those people being screened will not have the disease being sought?

MR. WHITLOCK: Objection to the form.

A. That may be true. It may be different here in Bennington. Because people have an elevated exposure to PFOA.

Q. Is it fair to say that medical screening in an asymptomatic population is often up against staggering odds?

MR. WHITLOCK: Objection to the form.

A. I don't know what staggering odds are. If this can be conducted with the purpose of finding cases early so that they can benefit from medical intervention at a stage where there is a substantial benefit to gain, I would say this is a good idea.

Q. I am not fussing when I say that, but there are a lot of ifs that are baked to that statement, true?

A. The way you state it, that is correct.

Q. Is it fair to say that if an asymptomatic population is being screened, it must involve many people to potentially benefit few?

MR. WHITLOCK: Objection to the form.

A. Again, that depends on the situation. It may be true on some situations but not on others.

Q. In assessing the utility of the medical monitoring program in an asymptomatic population, wouldn't one have to take its risks and benefits into account?

A. That would be normal.

Q. Is it fair to say that in a population of a thousand asymptomatic individuals being screened over a decade in an older population and even with a common cancer, only a few may be ten to twelve would be destined to die from a cancer?

A. That sounds reasonable but as a physician, I have to insist that every disease of subject, say, in among the residents in the zone of contamination is one we didn't do enough for to save.

Q. Is it fair to say that in assessing a medical

monitoring program in an asymptomatic population, the population being screened should be at a significantly high risk for the undiagnosed disease, that is, the disease should have a sufficiently high prevalence in the population?

A. Again, you are using the words significant and sufficient that are sort of, I don't understand what they mean, because you are speaking in general terms, but I understand what you are aiming at and I agree with that. Not necessarily with the specific way that you said it.

Q. Is it fair to say that in assessing medical monitoring program in an asymptomatic population, that the disease being screened for should have a sufficiently high prevalence in that population?

A. When you say sufficient, it is again a matter of consideration, and, you also, you continue to say the whole population is asymptomatic. That is very unlikely. So what we are dealing with here, we are dealing with an exposed population which must have an elevated risk. So this is why it is reasonable to consider medical monitoring to catch those cases that can be caught early so they can benefit from medical intervention.

Q. Yet, sitting here today, you cannot quantify what that elevated risk might be for any particular endpoint in terms of giving a relative risk point estimate or 95 percent confidence intervals around the point estimates, can you?

A. We are talking two things here. I am capable of doing that, but I can't do it right here on the spot and especially with the little information you give me.

Q. Well, I put no constraints on the scope of your report. Your report does not provide that information?

A. What information? What the relative risk is?

Q. Yes.

A. No, it does not because that is something that would depend on the circumstances like on the degree of exposure, duration, etcetera. That was not what I was asked to do. It is nothing that I would be able to do in a jiffy.

Q. Is it fair to say that in assessing a medical monitoring program in an asymptomatic population, one should consider the natural history of the disease and the evidence for an improved clinical outcome as a result of the medical monitoring for the given disease?

MR. WHITLOCK: Objection to the form.

A. What you refer to is the material that I have

been teaching myself. I can only agree. I would still comment on your insistence that it is a nonsymptomatic population. I don't think that is likely.

Q. The goal of the detecting disease early through screening is to improve the outcome when compared to the outcome of the same disease that is identified because the patient experiences symptoms and goes to see the doctor; is that correct?

A. Let me hear that again. There were several components.

Q. The goal of detecting disease earlier through screening is to improve the outcome when compared to the outcome of the same disease that is identified, because the patient experiences symptoms and then goes to see the doctor?

A. Compared to those that were identified through the screening, right, correct.

Q. So that's a correct statement?

A. It is.

Q. Early detection through screening should be known to have an impact on the natural history of the disease process, true?

A. Again, it depends on what you mean when you say natural history. I would say the course of the disease,

because you aim at identifying at early stage and then you want to impact that rather than waiting until the full blown clinical picture.

Q. The period of time between when a cancer starts growing and when it causes symptoms is when screening can catch the cancer; is that true?

A. That is at least the aim of screening.

Q. Is it fair to say that one reason there is a limited benefit to cancer screening is because there can be cancers that are missed by screening?

MR. WHITLOCK: Objection to the form.

A. It is part of the story that you can only miss some. That you can also detect some that were never meant to become a lethal cancer. So there are issues like that. It is a little more complicated than you stated.

Q. Aggressive, fast growing cancers are often missed by screening, aren't they?

A. That is one possibility. Yes.

Q. What population based cancer screening tests, if any, have ever been shown to reduce overall mortality?

A. I don't remember that by heart. I would refer to the National Cancer Society or CDC for that information.

Q. Are there any?

A. I'm sure.
Q. As a physician, do you subscribe to the proposition that health is not only a physical state of being, but is also a state of mind?
A. Well, the WHO definition does not say state of mind but it refers to mental health as well.
Q. Do you believe that mental health is part of a person's health status?
A. I believe so.
Q. Do you agree that as a physician, you must be careful not to undermine a person's state of health?
A. I don't think that is something every physician would consider doing. It certainly plays a role.
Q. Do you agree that in order for medical screening tests to be useful, their benefits must outweigh their risks?
A. I need to understand the word risk here. Are you referring to the risk, let's say, of a liver biopsy if that is what is considered necessary for the screening, is that the kind of risk?
Q. That could be a risk. There could be a risk of adverse psychosocial sequelae?
A. Fair enough. I agree that one has to take the whole range of possible risks into consideration as the

cost to the individual patients in addition to the advantages.

Q. Do you agree that to determine a test's usefulness, one should consider its specificity and sensitivity?

A. Yes.

Q. Do you agree that in the context of this case for a test to be useful, early detection must improve the outcome or the natural history of the disease?

A. The question is when you say must improve, we may not have that evidence available for a population like this. So it must at least judged to be beneficial.

Q. Do you agree that for a test to be useful, it must have a positive predictive value in a population at issue?

A. Yes. I would say so.

Q. Do you agree that knowledge of disease prevalence is a necessary component of positive predictive value?

A. Yes. But the problem is we may not know that exactly for a population. This may have to be decided on whatever information that there is available that can be applied.

Q. Is it fair to say that in assessing a medical monitoring program, one should consider the sensitivity,

specificity, and positive predictive value of proposed medical monitoring tests?

A. Yes. To the extent that's possible.

Q. Is it fair to say there may be serious consequences in the use of screening test with poor sensitivity or poor specificity?

MR. WHITLOCK: Counsel, for the record, we are getting beyond what we agreed upon here.

MR. WOLFF: I have not asked about specific tests like BUN or globulin or albumin, or alkolene, whatever it was. So I think we are not departing.

A. Could you repeat the question? I'm happy to answer.

Q. Sure.

Is it fair to say there may be serious consequences in the use of screening tests with poor sensitivity or poor specificity?

A. The question is in a word serious, what you mean by serious. Certainly there can be adverse questions. The circumstances will then define whether they can be considered serious.

Q. What difference is there if for the data required in determining the presence -- strike that.

What difference is there if any for the data

required in determining the prevalence of a health disturbance in a population as opposed to conducting a risk assessment for a health disturbance in a population?

A. I am a little confused about that. You are talking about risk assessment. Is that now a risk assessment for the PFOA or risk assessment for the procedure? I don't understand.

Q. Okay. In terms of an environmental exposure.

A. Okay. Like PFOA?

Q. Like PFOA.

A. Can you repeat the sentence?

Q. What difference is there if any for the data reuired in determining the presence of health disturbance in a population as opposed to conducting a risk assessment for a health disturbance in a population?

A. The first one descriptive epidemiology, you want to know how frequent it is; and the second one is analytic epidemiology where you are trying to relate that to particular suspecting.

Q. When you say how frequent it is in the population, are you referring there to its prevalence?

A. That would be the normal, that is, the occurrence

at a particular point. That would be the usual measure.

Q. In the context of a clinical or a laboratory test, what is false positive?

A. A false positive is one that indicates the presence of an abnormality where other evidence confirms or suggests that it is not present.

Q. A false positive is a false alarm, isn't it?

A. Yes. You could call it that.

Q. In the context of a clinical or a laboratory test, what is a false negative?

A. A false negative is one that is missed by the test despite the fact that other evidence, perhaps even later on documents that in fact is is present.

Q. What is overdiagnosis?

A. Overdiagnosis means that there are apparently included cases of false positives or that subject are being diagnosed without due justification.

Q. Is it fair to say that overdiagnosis is sometimes referred to as the detection of a cancer that is not destined to ever cause symptoms or death?

A. Right. I have seen literature on that for example with regard to breast cancer screening. It is an issue.

Q. Is it fair to say that overdiagnosis is the

diagnosis of a neoplasm that never would have clinical significance?

A. Well, it depends on the definition of whether you are, will, accept that a neoplasm has some consequence but not causing death. It depends on your definition of the situation. I agree with you in general terms.

Q. Is it fair to say that overdiagnosis is the identification of slow growing cancer that even if untreated would never cause symptoms or reduce survival because the screening test cannot distinguish between the abnormal appearing cells that would become cancerous from those that would never do so?

MR. WHITLOCK: Objection to the form.

A. You have just mentioned an example of that. That could be with regard to breast cancer. I agree that this is an actual problem that needs to be considered.

Q. A false positive test result effectively means that you are sending a healthy patient to a doctor for some type of follow-up; is that correct?

A. So type of?

Q. Follow-up.

A. Follow-up, because of a positive test. I assume that is the case.

Q. And a false negative test result, effectively

means that you are clearing a patient who should be referred to a doctor for some type of follow-up?

MR. WHITLOCK: Objection to form.

A. Right. That would be the proper explanation of that.

Q. What are the harms related to a false positive test result?

A. It depends on the circumstances.

Q. What are the harms related to a false positive test result for kidney cancer?

A. It could be a follow-up at the family doctor. If he also has a false positive, then there could be additional, therapeutic attempts on that kidney cancer, but I would assume that rather than the family physician referring the patient to radiation treatment or something dramatic, I would assume that there would be some additional steps so that the false alarm would be caught in time before the kidney was removed.

Q. Is it fair to say that another type of harm from a false positive is that the patient will become labeled with having a disease that is not present?

A. I don't quite know what you mean by saying label. Certainly, first of all, the patient can be scared himself or herself, and that could theoretically be some

sort of stigma. Talking about theory here.

Q. What are the harms related to a false negative test result?

A. I think we are going over the same ground again. Clearly, if worse comes to worse, the patient will unreasonably believe to be free of disease and may not take proper precautions potentially. So we are all trying to avoid false negatives clearly because we do want to do proper diagnosis at the proper time.

Q. That's because persons with false negative results may have delays in diagnosis and treatment; is that correct?

A. That could be the consequence.

Q. On the other hand, false positive results can result in followup testing that is uncomfortable, expensive, and potentially harmful?

A. Potentially. Yes.

Q. Is it fair to say that although screening may prevent the development of disease related morbidity and mortality, positive test results, both false positive and true positive may lead to interventions that could be unnecessary or even risky because of overdiagnosis and overtreatment?

A. That's a theoretical possibility.

Q. Is it fair to say the normal ranges for biomechanical tests are often based on the 95 percent confidence intervals in a normal healthy population, that is, although everyone is healthy by convention, values outside of the 2.5 percent lower and upper extremes are considered to be abnormal?

A. That is the standard practice in biochemistry.

Q. It is fair to say that ordering six blood tests in a normal, healthy individual yields only a 74 percent chance that all six tests will be normal? In other words, there is a 26 percent chance that one or more may be abnormal; is that true?

A. On the condition, and you are not expressing the circumstances properly. Excuse me. If the tests are independent, you are correct; but oftentimes when for example the doctor wants to diagnose a liver disease, the doctor will select six related liver tests and under those circumstances the calculation is not correct. It is an exaggeration.

Q. With respect to independent tests, when ordering twelve tests in a normal person, there is a 54 percent chance that all twelve will be normal and a 46 percent chance that one or more will be abnormal. Is that true?

A. Theoretically, that's true but I don't know of

any physicians who will prescribe twelve independent tests. Because you are always focused on something that is relevant to the patient's circumstances and it could be, you know, it PFOA exposure or it could be let's say yellow sclera that would indicate a liver problem.

Q. Do you know how many independent tests Doctor Ducatman is recommending as part of his medical monitoring program?

A. I have not counted them but I think from my reading he has selected an appropriate selection of tests.

Q. Do you know whether it is more than twelve or less than twelve?

A. I don't remember.

Q. Is it fair to say that simply ordering tests in healthy individuals or in the absence of clinical suspicion of a disease may result in many false positive tests that can lead to false alarms, anxiety, additional testing, and possible morbidity or mortality from subsequent testing or interventions?

MR. WHITLOCK: Objection to the form.

A. What you are saying is theoretically possible, but I doubt that it is likely in this case.

Q. Do you agree that some false positive tests can

be in excess of fifty percent such as CAT scan for sub-clinical malignancies?

A. I don't have any specific knowledge so I can't give you a proper answer.

Q. Would you agree that across the spectrum of screening tests for sub-clinical disorders, it is not uncommon for the false positive rate to be at least five percent?

A. I would think this is correct; but I don't have any detailed knowledge myself.

Q. Would you agree that for a screening test to be more likely than not predictive, the positive predictive value should be greater than 0.5 or in other words greater than fifty percent?

A. I can't comment on that. I don't have any specific knowledge to relate this to.

Q. If we assume that the false positive rate of a screening test for a sub-clinical disorder is at least five percent, in order for the positive predictive value to be greater than 0.5, the prevalence of the disorder in the population would have to be fifty out of a thousand, do you agree?

A. I have not done the math in my head. The numbers seem to be okay.

Q. Among all the potential disorders you opine on in your report, is there any single one of them where you would expect the PFA mediated incidents to be greater than 0.5 or in other words greater than fifty percent?

A. I mean, fifty percent in a prevalence?

Q. Yes.

A. Prevalence of at least fifty percent, I don't expect any of them will.

Q. You are no doubt familiar with the US Preventive Services Task Force; is that correct?

A. Yes. I am familiar with it; but I can't remember any quotes that I can relate to.

Q. Are you aware that the US Preventive Services Task Force was established by a congressional mandate and is comprised of an independent volunteer panel of sixteen national experts in prevention and evidence based medicine?

A. I don't remember. It sounds correct.

Q. Is it fair to say that the task force is generally considered to be a reputable and reliable part of the medical community?

A. I would assume so.

Q. Have you ever been a member of that task force?

A. No.

MR. WOLFF: Mark this as Exhibit Number 13.

(General types of harm for consideration was marked as Exhibit Number 13 for identification)

Q. Exhibit 13 is an excerpt from the US Preventive Services Task force Procedure Manual. Please turn with me to Page 43. The second paragraph in Section 6.6.2 under the heading of General Types of Harm for Consideration. Do you see that?

A. I see that.

Q. Do you agree with the proposition that harms of screening may include psychological harm from the labeling, the harms of diagnostic studies to confirm the presence of the condition, and overdiagnosis of screen protected conditions?

A. This is a fair statement. I can't disagree with that.

Q. Do you agree with the proposition that because screening and other preventive interventions are implemented in asymptomatic persons with the goal of preventing future disease, one should place a high priority on considering the harms of overdiagnosis and overtreatment whereby the preventive service has the unintended consequence of creating quote, diseases, close quote, that often leads to unnecessary and

ineffective treatment?

A. Right. You read this. It is a fair statement. It is not in my report though.

Q. Do you agree with the proposition that harms of early treatment and overdiagnosis may accrued a patient whose condition may never have come to clinical attention or for whom the arms of treatment initiated prior to routine clinical protection were different or occurred earlier and/or over longer period of time? In other words, these are harms of treatment that would not have occurred in the absence of screening.

A. These are some general statements that I can't disagree with.

Q. Please turn with me to Page 42. Section 6.6.1. Are you there?

A. I am there.

Q. Do you agree with the conceptual notion that screening is intended for asymptomatic individuals in order to prevent or delay future health problems?

A. That is reasonably stated. Yes.

Q. Do you agree with the proposition that the burden of proof, that the benefits exceed the harms prior to recommending implementation of screening or other preventive services is thus higher than it is for

diagnosis or treatment of symptomatic conditions?

MR. WHITLOCK: Objection to the form.

A. I think that's a reasonable statement. All I can say is, I understand and I agree.

Q. Isn't one of the fundamental precepts of preventive testing is that one should avoid doing more harm than benefit?

A. That's correct. I agree.

Q. Doesn't screening all comers in an asymptomatic population mean that you increase the number of false positives each of which comes with an obligation to follow-up?

MR. WHITLOCK: Objection to the form.

A. It is a theoretical question. I understand what you are saying. It is not related to my report, though.

Q. Why not, why do you say that it is not related to your report?

A. Because I did not assess the proposed biomonitoring program in any detail. I just assessed the general appropriateness of the proposal.

Q. What's the distinction that you are drawing? So I mean, Doctor Ducatman advanced a proposed medical monitoring program in his reports. You read those reports and you basically say at a high level, you are

giving those recommendations your stamp of approval; is that correct?

A. It is a stamp of approval in general terms. I have not evaluated those in detail, because that was not what I was asked to do. It says here in the first paragraph on Page 1, we have been through that, and so I have not essentially dissected his proposal and applied those principles. I would assume that he did. I think you should ask Doctor Ducatman about it.

Q. Let me take it from the other direction. While you did not dissect Doctor Ducatman's proposal, on the other side of the coin, you, yourself, have not created or proposed a particular medical monitoring program for PFOA exposed populations; is that correct?

A. That is correct, because I was not asked to do that.

Q. At any time?

A. I mean all I was asked to do is here in that paragraph on Page 1.

Q. I mean look, I don't want to, you know, beat around the bush. You have not done this with respect to any population anywhere in the world, have you?

A. Done what?

Q. You have not constructed, developed, specified, created a medical monitoring program for PFOA exposed

individuals?

A. I have not done that.

Q. I thought we were communicating, I just wanted to make sure?

A. One thing is you know the principles. One thing you know the risk assessment and the exposure related outcomes. I didn't consider that to be my task to do. That's why I am asking the way that I am.

MR. WHITLOCK: Counsel, can we go off the record?

MR. WOLFF: Sure.

THE VIDEOGRAPHER: The time is 3:34. We are off the record.

(Discussion off the record)

THE VIDEOGRAPHER: The time is 3:40. We are back on the record.

Q. Let me just see if I understand this. Is it your opinion that medical monitoring is warranted based on exposure to PFOA, but that you have not specifically undertaken to determine whether any particular tests or diseases for which Doctor Ducatman proposes to medically monitor or warranted?

A. I think that is correct interpretation.

Q. You prepared a case in chief, expert report in

support of the plaintiffs in PFOA litigation in New Hampshire; is that correct?

A. I did.

Q. That report is dated June 22, 2018; is that correct?

A. I believe that is the correct date.

Q. You prepared your case in chief report for the New Hampshire litigation approximately six weeks before your rebuttal report in this matter; is that correct?

A. That's correct. So they are quite similar.

Q. My question is, did you use your case in chief report in the New Hampshire litigation as a model for your rebuttal report in this matter?

A. I would not say I used it as a model, but I used my wordings here and there because the evidence that I am relying upon is the same and may have been updated with, some, you know, recent publications that came out after the June date.

Q. Do you agree the majority of your rebuttal report in this matter is the same as your case in chief report in the New Hampshire litigation?

A. I have not checked to which extent they are the same. I would say they are very similar because the situations are similar, but in that case there were no

rebuttals I needed to confront.

Q. Your case in chief report in New Hampshire was not prepared to rebut another expert's opinion?

A. That's correct.

Q. Your case in chief report in the New Hampshire litigation was not prepared to support any opinion by Doctor Ducatman in that matter, true?

A. I don't think he was involved in that case.

Q. He is not.

A. Okay. So that explains that.

Q. On Pages 44 and 45 of your report in this matter, you discuss diabetes; is that correct?

A. That's correct.

Q. Diabetes is not an endpoint that Doctor Ducatman is proposing to medically monitor for, is it?

A. I don't remember so; but it is worthwhile considering.

Q. You also discuss obesity in your report; is that correct?

A. That's correct.

Q. At the top of Page 55, you write that monitoring of body weight and BMI is essential in preventing cardiovascular disease recommendations for identifying subjects for possible obesity interventions have been

recently published; is that correct?

A. That's correct.

Q. Doctor Ducatman is not proposing that obesity be medically monitored as an endpoint, is he?

A. I don't remember, but you will see the references here are from 2018. So maybe he did not know the references at the time that he wrote this report.

Q. Monitoring BMI, monitoring body weight and monitoring for obesity is something that happens as a matter of routine medical care, virtually any time a patient walks into a clinical setting; is that true?

A. I don't know because there may be people not covered by this kind of service. I can't say that this is true in general.

Q. I don't mean to be flippant when I say this, but I must say from the moment I was born I was weighed and any other time I have ever stepped in a doctor's office, I am asked to takeoff my shoes by the nurse and get on the scale and be weighed. So is that your experience, too?

A. But I still seem to remember that Doctor Ducatman said that part of the medical monitoring was for the patient or the subject to step on the scale. So he is still saying this should be part of it and I agree.

Q. Stepping on the scale is no different than what most patients experience when they go in for a routine checkup, right?

A. If they do. So I have to side with Ducatman that he designed a program that I think is appropriate. Of course, height and weight is part of that. You cannot just exclude it because you think that people will go for a medical checkup because they may not.

Q. On Page 62, you also describe monitoring procedures for bladder cancer and prostate cancer; is that correct?

A. Right.

Q. Doctor Ducatman is not proposing medical monitoring for bladder cancer or for prostate cancer, is he?

A. I think you are correct. He is not.

Q. Well, why didn't you limit your opinions in your rebuttal report to the endpoints that Doctor Ducatman is proposing to medically monitor for?

A. It was because I was not asked to do that. I mean, if we were going to compare ideas and proposals for medical monitoring, I would have to share the references that I was familiar with and that I had picked up. Also, those after he completed his report

and we did not do that. It is clear that when I refer to that, let's say, Swedish study on prostate cancer, he may not have picked that up. And so we may deviate a little bit. That is only natural.

Q. Just to be fair and to state the obvious, the scope of your report goes beyond the scope of what Doctor Ducatman is proposing to medically monitor for; is that true?

A. That is not correct. My report focuses on the elevated exposure and elevated risks of what they are and that can of course be used as part of a discussion and decision on medical monitoring but that was not my task.

Q. What was your task?

A. To review the exposure associated risks of adverse health effects.

Q. Your rebuttal report was not targeted solely at the defense experts opinion, was it?

A. Not solely. Because I was looking at, I think you call it general causation. It was focused on what do we know about the adverse health effects.

Q. A general causation analysis was really the primary focus of your report?

MR. WHITLOCK: Objection to form.

A. We would have to ask a counsel about that. I was given the task as described here and clearly I spent more space on evaluating the strength of the evidence than I did on rebutting some report. I think I spent a page on each of them. That is really a very small component.

MR. WOLFF: Let's go off the record for a moment.

THE VIDEOGRAPHER: The time is 3:49. Going off the record.

(Discussion off the record)

THE VIDEOGRAPHER: Back on the record. The time is 3:50.

MR. WOLFF: Thank you. I have no further questions at this time.

CROSS EXAMINATION

BY MR. WHITLOCK:

Q. Doctor Grandjean, I just have one question for clarification purposes. Towards the end there of Mr. Wolff's questioning, there was several questions about the scope of your testimony in regards to the design of Doctor Ducatman's medical monitoring program. There was a question asked about your, the level of your review of the tests and the record will speak for

itself, but I believe disease endpoints as recommended by Doctor Ducatman in his medical monitoring program. I just want to be clear, on Pages 27 through 62, I believe, of your report, Section 8, which is titled Adverse Health Effects at Individual Endpoints, did you in that section of your report evaluate the epidemiological literature along with the toxicological evidence as you discussed throughout your deposition today in regards to the specific disease endpoints that Doctor Ducatman chose for his medical monitoring program?

MR. WOLFF: Objection to form.

A. I believe I covered the endpoints that he assessed and considered appropriate for his monitoring. I think I covered all of them.

MR. WHITLOCK: Thank you. That is my only question.

MR. WOLFF: We are done. Thank you.

THE VIDEOGRAPHER: The time is 3:52. We are going off the record. This is the end of today's deposition of Philippe Grandjean.

C E R T I F I C A T E

I, Philippe Grandjean, having read the foregoing transcript of my testimony, do hereby certify under the pains and penalties of perjury. The same contains a true and accurate record of my answers to the questions herein set forth, together with correction pages, if any, attached.

_____

PHILIPPE GRANDJEAN

Subscribed and sworn to before me

this ____ day of _____, 2018.

_____

NOTARY PUBLIC

C E R T I F I C A T E

Commonwealth of Massachusetts
Suffolk, ss.

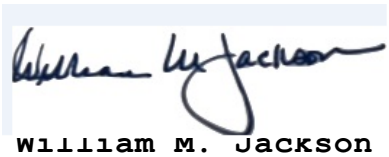
I, William M. Jackson, a notary public in and for the Commonwealth of Massachusetts, do hereby certify:

That Philippe Grandjean, the witness whose testimony is hereinbefore set forth, was duly sworn by me, and that such testimony is a true and correct transcription of my stenotype notes taken in the foregoing matter, to the best of my knowledge, skill and ability.

I FURTHER CERTIFY that the foregoing transcript is a true and correct record of the testimony given by the said witness at the time and place specified hereinbefore.

I FURTHER CERTIFY that I am neither a relative nor employee of nor counsel for any of the parties, nor am I financially interested directly or indirectly in the outcome this action.

IN WITNESS WHEREOF, I have hereunto set my hand and Notarial Seal this 13th day of November, 2018.



William M. Jackson
Notary Public

My Notary Commission expires: October 26, 2018.

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CASE: SULLIVAN VS. SAINT-GOBAIN PERFORMANCE

DEPOSITION DATE: OCTOBER 10, 2018

DEPONENT: PHILIPPE GRANDJEAN

PAGE	LINE(S)	CHANGE	REASON

_____

PHILIPPE GRANDJEAN

SUBSCRIBED AND SWORN TO BEFORE ME

THIS _____ DAY OF _____, 20___.

_____ _____

(NOTARY PUBLIC) MY COMMISSION EXPIRES:

f

Federal Rules of Civil Procedure

Rule 30

(e) Review By the Witness; Changes.

(1) Review; Statement of Changes. On request by the deponent or a party before the deposition is completed, the deponent must be allowed 30 days after being notified by the officer that the transcript or recording is available in which:

(A) to review the transcript or recording; and

(B) if there are changes in form or substance, to sign a statement listing the changes and the reasons for making them.

(2) Changes Indicated in the Officer's Certificate. The officer must note in the certificate prescribed by Rule 30(f)(1) whether a review was requested and, if so, must attach any changes the deponent makes during the 30-day period.

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